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OF THE
American Veterinary Medical Association
FORMERLY AMERICAN VETERINARY REVIEW

(Original Official Organ U. S. Vet. Med. Ass'n.)

H. Preston Hoskins, Secretary-Editor, 716 Book Building, Detroit, Mich.

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July, 1929

No. 1

THE CLASS OF 1929

Within the past few weeks 143 new names have been inscribed on the roll of the veterinary profession in the United States and Canada. To these young men the American Veterinary Medical Association extends greetings. Some have already applied for membership in the A. V. M. A.—a good sign. The majority have already had contact with the national organization through the nine student chapters of the A. V. M. A., in as many of the veterinary colleges. Just a friendly word of advice to these 143 neophytes, from one who has been in a wonderful position to observe both the good and bad points of veterinarians, under circumstances of many kinds.

In entering the veterinary profession you have assumed an obligation to those men already in the ranks, many of whom have contributed much to place the profession on the high plane that it occupies today. To use common parlance, it is up to you to play the game according to the rules. Some of these rules are on the books. Others are unwritten. It might be more appropriate to refer to the latter as traditions. It is up to you to uphold them. Some would lead you to believe that ethics have been relegated to the limbo of the forgotten past. Not so. Our code of professional conduct is just as reliable a standard today as it ever was. Tie to it. Just because a veterinarian occasionally renounces this standard is no reason to believe that our code has

been broken down. These infractions are the exceptions and not the rule.

You are entering the profession at a most auspicious time. There are opportunities on every hand. Splendid positions have gone begging the past year, awaiting the right men to fill them. The public, slowly but surely, is beginning to recognize the value of the service rendered by veterinarians. The profession has been accused of hiding its light under a bushel. Perhaps it has, but which is better, an accusation of this kind, or one to the effect that we are a group of blatant advertisers? In the quotation, "By the work one knows the workman," there is something refreshing to contemplate. It is a source of deep satisfaction to feel that the profession is looked upon as being modest rather than offensively self-assertive. Help guard this good reputation.

Just as soon as possible, affiliate with your professional associations, local, state and national. When you do, be more than a name on the membership roll. Attend as many meetings as you can. When the secretary asks you to contribute to the program of a meeting, do so. Subscribe to your professional journals and read them. Keep your library up to date. Your purchase of each new book lends encouragement to others to write more books. We must have our literature. Books are just as important parts of a veterinarian's armamentarium as his instruments, his drugs, or his operating-table. Take an interest in civic affairs. The broadening influence of the contacts you make in church, school, business and civic organizations will be wonderfully helpful in making yourself a good citizen as well as a successful veterinarian.

Again, we greet you, the class of 1929.

Drive to Dynamic Detroit.

Two New Student Chapters

The Executive Board recently approved the organization of two additional student chapters of the A. V. M. A. The first was organized at the Georgia State College of Agriculture. Thirty regularly enrolled students in the Division of Veterinary Medicine applied for a charter. The most recently organized student chapter is at the Texas Agricultural and Mechanical College. At this institution fifteen regularly enrolled students in the School of Veterinary Medicine filed the petition. We now have nine student chapters organized.

COMPLETING OUR SURVEY

This month we are concluding our statistical studies of A. V. M. A. membership strength in the various states. There are thirteen states in the list considered this month and the number of A. V. M. A. members in these states at the present time varies from ten, in the case of New Mexico, to thirty, in Louisiana. Practically all of these states show losses in membership strength as compared with the year 1923, which has been the basis of our comparisons of other states previously reported upon. With the exceptions of four states, the shrinkage in membership exceeds 20 per cent and runs as high as 44 per cent.

The standing of Mississippi has been helped tremendously through the filing of eight applications for membership during the past month. We have to thank Dr. Hartwell Robbins, B. A. I. inspector-in-charge of tick eradication in the Bayou State, for securing these applications.

Louisiana shows a loss of just exactly one-third of her membership strength during the past six-year period. There have been indications of an improvement in the veterinary situation in Louisiana the past year and it is to be hoped that this will be reflected in an increase in interest in the A. V. M. A. upon the part of the veterinarians in the Pelican State.

Georgia is another state that shows a rather heavy loss. The number of approved veterinarians in the Cracker State, according to the latest information, is 105. Of the thirteen states in this group, Georgia has the largest number of approved veterinarians and at the same time the lowest percentage of members of the A. V. M. A.

Idaho, with a loss of only two members, has improved her position wonderfully through the efforts of our resident secretary for that State, Dr. W. A. Sullivan. There are forty approved veterinarians in Idaho and, with twenty-nine members in the A. V. M. A., Idaho shows up very well on a percentage basis.

Tennessee is another state with a small loss of five. However, with ninety approved veterinarians in the Big Bend State and only twenty-seven A. V. M. A. members, Tennessee does not show up so well.

Vermont is the only state in the list to show a gain during the past six years. Vermont had 24 members in 1923 and has just one more at the present time. Just a trifle over 50 per cent of

the approved veterinarians in the Green Mountain State are affiliated with the A. V. M. A.

South Carolina has a small loss of five, but this is almost 20 per cent of her A. V. M. A. membership strength six years ago. South Carolina shows up very well, however, on a percentage basis, over 70 per cent of the approved veterinarians in the Palmetto State being members of the A. V. M. A.

West Virginia shows a loss of seven members, which is almost 25 per cent. The twenty-two members of the A. V. M. A. credited to West Virginia at the present time represent considerably less than one-third of the number of approved veterinarians in the Panhandle State.

Montana shows a loss of six, almost one-fourth of the membership strength in 1923. The latest information shows that there are forty-eight approved veterinarians in the Stub Toe State and twenty members of the A. V. M. A. This is about the average for the country as a whole.

Arkansas shows a comparatively heavy loss of eleven. Resident Secretary Bux has been putting forth valiant efforts to improve the standing of the Bear State during recent months and the loss shown by Arkansas may be reduced by the end of the year.

Maine shows a very heavy loss, almost one-half her A. V. M. A. membership strength in 1923. The Pine Tree State is charged with forty-nine approved veterinarians and the thirteen A. V. M. A. members in Maine bring the percentage for that state to 27, which is considerably below the average.

Utah shows a loss of four and this represents a shrinkage of over 20 per cent during the six-year period. With thirty-two approved veterinarians on the list, Utah has about the average A. V. M. A. membership strength for the entire country.

At this point it might be well to direct attention to the fact that the two states just considered, Maine and Utah, with thirteen and fourteen members respectively, are the only two states in the Union with all members having their dues paid up in full, at the time these figures were compiled.

New Mexico shows a relatively heavy loss of seven members, representing over one-third of the number on the books in 1923. However, there are only fifteen approved veterinarians in the State according to most recent information and New Mexico shows up very well on a percentage basis.

<i>State</i>	<i>1923</i>	<i>1929</i>	<i>Change</i>
Mississippi.....	37	32	— 5
Louisiana.....	45	30	—15
Idaho.....	31	29	— 2
Georgia.....	43	28	—15
Tennessee.....	32	27	— 5
Vermont.....	24	25	+ 1
South Carolina.....	28	23	— 5
West Virginia.....	29	22	— 7
Montana.....	26	20	— 6
Arkansas.....	29	18	—11
Utah.....	18	14	— 4
Maine.....	24	13	—11
New Mexico.....	18	11	— 7

This survey could not be complete without a word about Canada. In 1923 we had 173 Canadian members, compared with 161 at the present time. This loss of twelve members does not tell the whole story, however. The A. V. M. A. met in Montreal in 1923 and this resulted in a big increase in membership from the provinces, reflected in the report for 1924, when Canada was credited with 222 members. This number has been steadily decreasing, to 161 at present, or 61 below the peak.

DETROIT HOTELS

HOTEL	DISTANCE FROM HEADQUARTERS	ROOMS	RATES	
			SINGLE WITH BATH	DOUBLE WITH BATH
Book-Cadillac	(Official headquarters)	1200	\$3 Up	\$5 Up
Statler	3 blocks	1000	\$3 Up	\$5 Up
Tuller	4 blocks	800	\$2.50 Up	\$5 Up
Detroit-Leland	3 blocks	700	\$3 Up	\$5 Up
Fort Shelby	3 blocks	900	\$3.50 Up	\$5 Up
Barlum	3 blocks	810	\$2.50 Up	\$4 Up
Wolverine	6 blocks	500	\$2.50 Up	\$4.50 Up

CAMP SITE AT DETROIT

The Local Committee on Arrangements has been assured that camping facilities will be provided at the State Fair Grounds, for veterinarians and their families attending the Detroit meeting. Protection of camping equipment will be provided by the state police during the time that it is necessary to be down town attending the various sessions. The State Fair Grounds are located on North Woodward Avenue, about five miles from the Book-Cadillac Hotel, the official headquarters. Ample time for getting down town should be allowed on account of the heavy traffic on Woodward Avenue.

OUTLINE OF PROGRAM**MONDAY, AUGUST 12, 1929**

- MORNING** . . . Committee meetings.
AFTERNOON . . Meeting of Executive Board.
EVENING . . . Conference of accredited delegates from state, provincial and territorial veterinary associations.

TUESDAY, AUGUST 13, 1929

- MORNING** . . . Registration. Opening session, 10 a.m., Grand Ball Room. Addresses of welcome. Response. President's address. Ladies attend.
AFTERNOON . . General session, 2 p.m., Grand Ball Room. Reports of Executive Board and officers. Ladies attend card party and tea, Crystal Ball Room, 2:30 p.m.
EVENING . . . Meeting of college alumni groups, 6 p.m. President's reception and dance, 8 p.m.

WEDNESDAY, AUGUST 14, 1929

- MORNING** . . . Sectional meetings, 9 a.m. Papers. Ladies go on trip to Bob-lo. Leave Book-Cadillac at 8:15 a.m.
AFTERNOON . . General session, 2 p.m., Grand Ball Room. Reports of Executive Board and committees.
EVENING . . . Banquet, 6:30 p.m.

THURSDAY, AUGUST 15, 1929

- MORNING** . . . Sectional meetings, 9 a.m. Papers. Ladies go on shopping tour, 9 a. m.
AFTERNOON . . General session, 2 p.m., Grand Ball Room. Reports of Executive Board and committees. Women's Auxiliary meets, Crystal Ball Room, 2:30 p.m.
EVENING . . . General session, 8 p.m., Grand Ball Room. Papers. Installation of officers. Ladies attend theatre party at the "Michigan," 7 p.m.

FRIDAY, AUGUST 16, 1929

- MORNING** . . . Clinic, State Fair Grounds, 8:30 a.m. Ladies go on sight-seeing trip, thence to the State Fair Grounds for a joint picnic luncheon with the gentlemen, at 12:15 p.m.
AFTERNOON . . Clinic continued, 1:30 p.m.

SATURDAY, AUGUST 17, 1929

- MORNING** . . . Automobile trip to Parkedale Farm, Rochester, and Ingleside Farm, Mt. Clemens.

PAPERS

SECTION ON EDUCATION AND RESEARCH

1. The Department of Animal Pathology at Cambridge University—Dr. E. L. Stubbs, University of Pennsylvania, Philadelphia, Pa.
2. The Minimum Lethal Dose of Hog Cholera Virus—Dr. C. N. McBryde, Biochemic Division, U. S. Bureau of Animal Industry, Ames, Iowa.
3. A Double Intradermal Test for Infectious Abortion—Drs. B. H. Edgington and Alvin Broerman, State Department of Agriculture, Reynoldsburg, Ohio.
4. A Comparison of the Agglutination and Complement-fixation Tests for the Detection of *Brucella Abortus* Infection—Dr. Alexander Zeissig and Harriet L. Mansfield, Cornell University, Ithaca, N. Y.
5. Pathogenicity of Abortion Vaccine for Guinea Pigs—Drs. J. P. Torrey and E. T. Hallman, Michigan State College, East Lansing, Mich.
6. The Pathogenic Significance of Spirochaetes in Some Well-known Pathological Conditions of Animals (Illustrated)—Dr. J. A. Howarth, University of California, Davis, Calif.
7. The Fate of Acid-fast Bacilli Introduced into the Peritoneal Cavity of Guinea Pigs (Illustrated)—Dr. W. A. Hagan and Harriet L. Mansfield, Cornell University, Ithaca, N. Y.
8. The Relation of *Bacterium Abortus* to Fistula and Poll-evil of Horses—Dr. C. P. Fitch, University of Minnesota, St. Paul, Minn.
9. Coccidiosis in Swine and Studies on *Eimeria Zurni* and *E. Debliecki*—Drs. H. E. Biester and Chas. Murray, Iowa State College, Ames, Iowa.
10. Investigations of Canine Diseases with Special Reference to Rabies—A Preliminary Report—Drs. W. J. Lentz, University of Pennsylvania, Philadelphia, Pa.; M. F. Barnes, Pennsylvania Bureau of Animal Industry, Harrisburg, Pa., and A. N. Metcalf,
11. The Use of Salts in the Production of Blackleg Filtrate and Bacterin—Dr. Joseph P. Scott, Kansas State Agricultural College, Manhattan, Kans.

SECTION ON SANITARY SCIENCE AND FOOD HYGIENE

1. Legal Aspects of Tuberculosis Eradication—Dr. A. E. Wight, Chief, Tuberculosis Eradication Division, U. S. Bureau of Animal Industry, Washington, D. C.
2. Rabies Vaccine in the Control of Rabies—Dr. H. W. Schoening, Pathological Division, U. S. Bureau of Animal Industry, Washington, D. C.
3. The Control of Rabies in Pennsylvania—Dr. T. E. Munce, Director, Pennsylvania Bureau of Animal Industry, Harrisburg, Pa.
4. Public Health Aspect of Undulant Fever—Dr. W. W. Lee, Epidemiologist, Indiana State Board of Health, Indianapolis, Ind.
5. Military Meat and Dairy Hygiene in Relation to Army Health—Lt. Col. R. C. Musser, Veterinary Corps, U. S. Army, Washington, D. C.
6. Municipal Meat and Milk Inspection Work—Dr. H. B. Mitchell, Lancaster, Pa.

SECTION ON GENERAL PRACTICE

1. The Practical Application of Veterinary Biologics—Dr. M. J. Harkins, University of Pennsylvania, Philadelphia, Pa. Discussion by Dr. W. A. Axby, Harrison, Ohio.
2. The Pennsylvania Plan as Applied in Fourteen Herds—Drs. B. Scott Fritz and M. F. Barnes, Pennsylvania Bureau of Animal Industry, Harrisburg, Pa.
3. Anemia in Young Pigs (Illustrated)—Dr. R. A. Craig, Purdue University, Lafayette, Ind. Discussion by Mr. L. P. Doyle, Purdue University, Lafayette, Ind.
4. Equine Breeding Problems—Dr. Cassius Way, New York, N. Y.
5. Some Common Ailments of Sheep—Dr. F. E. Stiles, Battle Creek, Mich.
6. Recent Progress in our Knowledge of Milk Fever—Dr. P. A. Fish, Cornell University, Ithaca, N. Y. Discussion by Dr. C. J. Marshall, University of Pennsylvania, Philadelphia, Pa.
7. The Treatment of Tetanus in Horses—Dr. W. J. R. Fowler, Ontario Veterinary College, Guelph, Ont., Canada. Discussion by Dr. E. E. Patterson, Detroit, Mich.

8. An Experimental Study of Drugs Stimulating the Motility of the Ruminant Stomach—Dr. Roger S. Amadon, University of Pennsylvania, Philadelphia, Pa.

SECTION ON SMALL ANIMALS

1. Routine Microscopic Fecal Examinations in Small Animal Practice—Dr. E. R. Steel, Kansas City, Mo.
2. Cesarean Section—Dr. F. F. Parker, Des Moines, Iowa.
3. Diseases of the Kidney—Dr. Floyd Perrin, Lincoln, Nebr.
4. Foxes Whelped under Observation in Maternity Hospitals—Dr. J. A. Allen, Winnipeg, Man.

SECTION ON POULTRY

1. The Veterinarian—An Economic Factor in the Poultry Production Business—Dr. A. D. Goldhaft, Vineland, N. J.
2. Some Aspects of Fowl Pox and Its Control—Dr. F. R. Beaudette, New Jersey Agricultural Experiment Station, New Brunswick, N. J.
3. Federal Inspection of Live and Dressed Poultry—Dr. L. D. Ives, Bureau of Agricultural Economics, New York, N. Y.
4. Some Studies on Bang Disease in Chickens—M. W. Emmel and Dr. I. F. Huddleson, Michigan State College, East Lansing, Mich.

GENERAL SESSION

1. Liver Flukes (Motion picture)—Dr. J. N. Shaw and Dr. B. T. Sims, Oregon Agricultural College, Corvallis, Ore.
2. The 1929 Outbreak of Foot-and-Mouth Disease in California—Dr. John R. Mohler, Chief, U. S. Bureau of Animal Industry, Washington, D. C.
3. How to Handle Foxes (Motion picture)—Dr. J. E. Shillinger, Bureau of Biological Survey, Washington, D. C.
4. Problems in Veterinary Parasitology in the United States—Dr. M. C. Hall, Chief, Zoological Division, U. S. Bureau of Animal Industry, Washington, D. C.

***In the year of our Lord, 1929, and of the A. V. M. A., 66,
you can combine business and a pleasurable vacation.***

Detroit, August 13-16.

APPLICATIONS FOR MEMBERSHIP

For the second consecutive month, the number of applications to be given first listing in the JOURNAL has reached the century mark. The number of applications listed this month and last exceeds the total of applications completed during the entire year, 1926.

Texas was the "big noise" the past month, as will be noted by glancing at the following list. The Lone Star State came across with 21 applications, bringing her total for the year up to 29, and incidentally moving up from eleventh place to fifth, passing Indiana, Virginia, Ohio, Kansas, New York and Iowa.

Pennsylvania contributes 13 more. California comes up with an even dozen, Illinois and Iowa each with seven and Kansas, Michigan, Mississippi, Montana and South Dakota with three apiece.

The applications listed this month bring the total for the year (since the Minneapolis meeting) within a few of the 500 mark, thereby breaking the record established last year.

Do not forget the plan under which applications are now handled. The following is from the By-laws:

Application for membership shall be made upon a blank furnished by the Association, in the handwriting of the applicant, and must be endorsed by two members of the Association in good standing, one of whom must be a resident of the state, province or territory in which the applicant resides. Application must be accompanied by the membership fee of \$5.00 and dues pro rata for the balance of the fiscal year current, as stated on the application blank. Application must be filed with the Secretary and must be examined by him for correctness and completeness as far as available information will allow. After such approval by the Secretary, the latter will cause to be published in the official JOURNAL, as soon thereafter as possible, said application and name and address of applicant, college and year of graduation, and names of vouchers. If no objections shall be filed with the Secretary, as against the applicant being admitted to membership in the Association, his name shall again be listed in the next issue of the JOURNAL, and if no objections shall have been filed within thirty days after the second publication of the name of the applicant, he shall automatically become a member and shall be so enrolled by the Secretary, and membership card issued. If any objections be filed against any applicant, either on first or second notice, said application will be referred to the Executive Board for consideration.

FIRST LISTING

BAILEY, WILLIAM WESTLEY	Box 1092, Red Lodge, Mont.
D. V. M., Iowa State College, 1929	
Vouchers: C. H. Covault and W. F. Guard.	
BARNEY, JOSEPH CLEMENT	Magnolia, Mass.
V. M. D., University of Pennsylvania, 1920	
Vouchers: E. Heiny and Hartwell Robbins.	

- BARRY, JAMES R.** D. V. M., Iowa State College, 1929
Vouchers: C. H. Covault and W. F. Guard. Wonebec, Wis.
- BERETTA, EDWARD H** D. V. M., Iowa State College, 1929
Vouchers: C. H. Covault and W. F. Guard. Cresco, Iowa
- BLACKBURN, HERBERT LAWRENCE** 713 N. Main, Fort Worth, Texas
D. V. M., Cincinnati Veterinary College, 1912
Vouchers: N. F. Williams and W. R. McCuiston.
- BOLIN, FONSOE M** D. V. M., Iowa State College, 1929
Vouchers: C. H. Covault and W. F. Guard. Turtle Lake, Wis.
- BOTT, THOMAS L.** D. V. M., Grand Rapids Veterinary College, 1912
Vouchers: C. H. Clark and B. J. Killham. Coldwater, Mich.
- BOYCE, HAROLD J.** 610 Sandusky St., Kansas City, Kans.
D. V. M., Kansas City Veterinary College, 1917
Vouchers: N. L. Townsend and C. B. Clement.
- BOYD, BENJ. W.** Box 462, San Antonio, Texas
D. V. S., Kansas City Veterinary College, 1907
Vouchers: N. F. Williams and W. R. McCuiston.
- BOYER, THEODORE ROSS** 1663 21st Ave., San Francisco, Calif.
D. V. M., San Francisco Veterinary College, 1917
Vouchers: Joseph M. Arburua and M. J. O'Rourke.
- BROOKS, CLARENCE SHEPARD** 304 Mapleton Ave., Hollister, Calif.
D. V. M., San Francisco Veterinary College, 1915
Vouchers: A. C. Rosenberger and E. H. Barger.
- BROWN, WILBUR L.** 531 5th St., Fowler, Calif.
D. V. M., Colorado Agricultural College, 1917
Vouchers: A. C. Rosenberger and W. L. Curtis.
- CHANDLER, GUY D.** 4160 Ellis Ave., Chicago, Ill.
V. M. D., Indiana Veterinary College, 1911
Vouchers: Robert J. Robertson and H. Busman.
- CHAPPELL, ROBERT FORREST** Navasota, Texas
D. V. M., Southwestern Veterinary College, 1916
Vouchers: N. F. Williams and W. R. McCuiston.
- COFFLAND, W. T.** 895 Vista Ave., Pasadena, Calif.
D. V. M., McMillip Veterinary College, 1916
Vouchers: W. L. Curtis and J. P. Bushong.
- COLE, ALONZO B.** New Mulford, Pa.
B. V. Sc., Ontario Veterinary College, 1910
Vouchers: Peter N. McNeal and J. B. Reidy.
- COOKE, WM. A. JR.** 2057 Parrish St., Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1909
Vouchers: Wm. J. Lentz and G. A. Dick.
- COTE, FRANK J.** 300 Eramosa Road, Guelph, Ont.
B. V. Sc., Ontario Veterinary College, 1926
Vouchers: C. D. McGilvray and R. A. McIntosh.
- COVINGTON, C. R.** 701 Wheat Bldg., Fort Worth, Texas
D. V. M., Alabama Polytechnic Institute, 1921
Vouchers: N. F. Williams and W. R. McCuiston.
- EASTMAN, DONALD A.** 864 A Ave., Cedar Rapids, Iowa
D. V. M., Iowa State College, 1922
Vouchers: Grant B. Munger and John B. Bryant.
- EDELMAN, FRANK J.** 151 Main St., Bath, Pa.
D. V. M., Chicago Veterinary College, 1916
Vouchers: J. O. Reed and T. E. Munce.

- ELLIOTT, JOHN WILFRED 310 South 2nd St., Aberdeen, S. Dak.
V. S., Ontario Veterinary College, 1890
Vouchers: M. W. Ray and C. C. Lipp.
- ERVIN, WILLIAM J. 117 Beechwood Ave., Pawtucket, R. I.
M. D. C., Chicago Veterinary College, 1911
Vouchers: Joseph S. Barber and J. M. Armstrong.
- EWERS, JAMES E. 300 State House, Phoenix, Ariz.
D. V. S., Kansas City Veterinary College, 1910
Vouchers: M. Shipley and J. C. McGrath.
- EWERS, SMITH V. c/o Arizona Packing Co., Phoenix, Ariz.
D. V. S., Kansas City Veterinary College, 1909
Vouchers: M. Shipley and J. C. McGrath.
- FARRELL, JOSEPH MILTON 1509 Speedway, Wichita Falls, Texas
D. V. M., Southwestern Veterinary College, 1916
Vouchers: N. F. Williams and W. R. McCuiston.
- FICKENSHER, HARRY J. 1534 East 67th Place, Chicago, Ill.
M. D. C., Chicago Veterinary College, 1911
Vouchers: Robert J. Robertson and H. Busman.
- FITZPATRICK, DENNIS B. Falfurrias, Texas
V. M. D., University of Pennsylvania, 1893
Vouchers: N. F. Williams and W. R. McCuiston.
- GAW, HUGH 46 Barrows St., N. Attleboro, Mass.
D. V. S., McGill University, 1903
Vouchers: Joseph S. Barber, J. M. Armstrong and H. W. Jakeman.
- GEICK, WALTER L. G. 709 Fowler St., Waterloo, Iowa
D. V. M., St. Joseph Veterinary College, 1915
Vouchers: J. E. Shelton and A. L. Blake.
- GREEN, FRED B. Lufkin, Texas
D. V. M., McKillip Veterinary College, 1916
Vouchers: N. F. Williams and W. R. McCuiston.
- GREGORY, RECE CORNELL Georgetown, Texas
D. V. M., St. Joseph Veterinary College, 1922
Vouchers: N. F. Williams and W. R. McCuiston.
- GRIFFEY, JAMES HOMER 16 Highland Ave., National City, Calif.
D. V. M., San Francisco Veterinary College, 1917
Vouchers: L. K. Knighton and A. P. Immenschuh.
- GROVER, CAPT. SAWYER ADELBERT Fort Howard, Md.
D. V. M., Kansas City Veterinary College, 1914
D. V. M., McKillip Veterinary College, 1915
Vouchers: R. A. Kelser and R. C. Musser.
- HARRINGTON, CHARLES F. 933 East Fifth Ave., Mitchell, S. Dak.
M. D. C., Chicago Veterinary College, 1911
Vouchers: G. W. Cronen and J. O. Wilson.
- HARTNELL, BERTON THOMAS Stacyville, Iowa
D. V. M., Chicago Veterinary College, 1912
Vouchers: Grant B. Munger and Chris. E. Juhl.
- HAWKINS, J. T. Tyler, Texas
D. V. M., St. Joseph Veterinary College, 1920
Vouchers: N. F. Williams and W. R. McCuiston.
- HODGES, W. ROSS Ranger, Texas
D. V. M., Kansas City Veterinary College, 1912
Vouchers: N. F. Williams and W. R. McCuiston.
- HOVER, EUGENE Box 837, Reno, Nevada
D. V. M., Colorado Agricultural College, 1918
Vouchers: Edward Records and Warren B. Earl.
- HYLTON, FLOYD D. Box 90, Capitol Station, Helena, Mont.
D. V. S., Colorado Agricultural College, 1910
Vouchers: W. L. Carson and G. H. Ehlers.

- KINTNER, CAPT. JOHN H. Fort Sill, Okla
V. M. D., University of Pennsylvania, 1917
Vouchers: R. A. Kelser and R. C. Musser.
- KITCHEN, FRANK ELLIOTT Greenville, S. C.
D. V. S., Kansas City Veterinary College, 1916
Vouchers: W. A. Barnette and John H. Morse.
- LANHAM, ERNEST FLOYD Box 320, Amarillo, Texas
D. V. S., Kansas City Veterinary College, 1911
Vouchers: N. F. Williams and W. R. McCuiston.
- LEWIS, ALLAN JEFFERSON Haskell, Texas
M. D. C., Chicago Veterinary College, 1910
Vouchers: N. F. Williams and W. R. McCuiston.
- LINDLEY, A. T. Box 101, Winters, Texas
D. V. M., Arkansas Veterinary College, 1917
Vouchers: N. F. Williams and W. R. McCuiston.
- LUKENS, WILLIAM M. 5021 Mervine St., Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1929
Vouchers: G. A. Dick and R. S. Amadon.
- MCCAHOE, JAMES VINCENT San Marino Ave., Bryn Mawr, Pa.
V. M. D., University of Pennsylvania, 1929
Vouchers: J. Allyn Rogers and G. A. Dick.
- MCCRILLIS, HAROLD L. South Union and Park Ave., Des Moines, Iowa
D. V. M., Iowa State College, 1929
Vouchers: C. H. Covault and W. F. Guard.
- MAAS, EDWARD E. Columbia, Miss.
D. V. M., Kansas City Veterinary College, 1917
Vouchers: E. Heiny and Hartwell Robbins.
- MACK, JOSEPH PETER Starke, Fla.
D. V. S., New York University, 1917
Vouchers: Thurman W. Cole and Jean V. Knapp
- MARTIN, ARTHUR DUNLAP 1817 W. Pershing Rd., Chicago, Ill.
D. V. M., Indiana Veterinary College, 1915
Vouchers: R. A. Kelser and R. C. Musser.
- MERICLE, ROBERT B. Blue Earth, Minn.
D. V. M., Iowa State College, 1929
Vouchers: C. H. Covault and W. F. Guard.
- MERRILL, WINSLOW EATON 84 Burlington Ave., Wilmington, Mass.
V. M. D., University of Pennsylvania, 1929
Vouchers: E. T. Booth and G. A. Dick.
- MORRETTE, EDNOR MICHAEL CYRUS 217 S. Hanover St., Carlisle, Pa.
D. V. S., United States College of Veterinary Surgeons, 1910
Vouchers: Ray O. Whipple and T. E. Munce.
- MOUST, CLARK JAY Red Lion, Pa.
D. V. M., Grand Rapids Veterinary College, 1917
Vouchers: Ray O. Whipple and T. E. Munce.
- MUFFLY, JAMES ALBERT Watsontown, Pa.
V. M. D., University of Pennsylvania, 1929
Vouchers: E. P. Althouse and G. A. Dick.
- MUIR, GEOFFREY SPICER Wellesley, Ont., Canada
B. V. Sc., Ontario Veterinary College, 1928
Vouchers: C. D. McGilvray and R. A. McIntosh.
- MURPHY, GEORGE H. 1308 West 68th St., Chicago, Ill.
D. V. M., St. Joseph Veterinary College, 1920
Vouchers: Robert J. Robertson and H. Busman.
- MURRAY, FRED A. 910 East 6th St., Austin, Texas
D. V. M., A. & M. College of Texas, 1920
Vouchers: N. F. Williams and W. R. McCuiston.

- MURTY, BYRON C. Parkston, S. Dak.
D. V. M., Grand Rapids Veterinary College, 1915
Vouchers: C. C. Lipp and F. M. McConnell.
- NEVIN, LT. STANLEY M. c/o 2nd Medical Regiment, Fort Sam Houston, Texas
D. V. M., State College of Washington, 1925
Vouchers: R. A. Kelser and R. C. Musser.
- NILSON, WALTER L. Veterinary Division, Iowa State College, Ames, Iowa
D. V. M., Iowa State College, 1929
Vouchers: C. H. Covault and W. F. Guard.
- NORDSTROM, HAROLD Wausa, Nebr.
D. V. M., Iowa State College, 1929
Vouchers: C. H. Covault and W. F. Guard.
- NORRIS, FRED MARTIN 909 G St., Reedley, Calif.
M. D. C., Chicago Veterinary College, 1909
Vouchers: W. L. Curtis and A. C. Rosenberger.
- NUGENT, THOMAS F. 210 East Grant St., New Castle, Pa.
B. V. Sc., Ontario Veterinary College, 1909
Vouchers: T. E. Munce and Wm. Brod.
- O'MALLEY, GERALD F. 93 Summit St., Clinton, Mass.
D. V. M., Ohio State University, 1923
Vouchers: H. W. Jakeman and Thomas A. Doyle.
- OWEN, EDWARD RANDALL Lawton, Pa.
B. V. Sc., Ontario Veterinary College, 1916
Vouchers: T. E. Munce and Peter N. McNeal.
- PATTON, L. E. Sidney, Mont.
D. V. M., Ohio State University, 1911
Vouchers: Hadleigh Marsh and W. J. Butler.
- PECK, MARLAIN AUSTIN 713 North Main St., Fort Worth, Texas
D. V. S., Kansas City Veterinary College, 1904
Vouchers: N. F. Williams and W. R. McCuiston.
- PENNELL, BENJAMIN NIXON 277 Williams St., New London, Conn.
D. V. S., New York-American Veterinary College, 1906
Vouchers: Geo. E. Corwin and Edwin Laitinen.
- PETERSON, ELLIS 1914 L St., Sacramento, Calif.
M. D. V., Harvard University, 1897
Vouchers: David F. Fox and W. L. Curtis.
- RAGAN, GEORGE WASHINGTON 1308 Monroe St., Wichita Falls, Texas
D. V. M., Southwestern Veterinary College, 1915
Vouchers: N. F. Williams and W. R. McCuiston.
- RANKIN, FAY G. Foot of 7th St., Astoria, Ore.
D. V. M., Colorado Agricultural College, 1928
Vouchers: William H. Lytle and Thomas B. Carter.
- RAWLEY, WALTER F. Box 33, Palestine, Texas
D. V. S., Kansas City Veterinary College, 1910
Vouchers: N. F. Williams and W. R. McCuiston.
- REY, GEO. S. Route 2, Box 510, Visalia, Calif.
D. V. M., San Francisco Veterinary College, 1908
Vouchers: W. L. Curtis and Joseph M. Arburua.
- RIEDEL, CAPT. PHILIP H. U. S. Army, Fort Jay, N. Y.
D. V. M., Indiana Veterinary College, 1911
Vouchers: R. C. Musser and R. A. Kelser.
- RYNKIEWICZ, JOSEPH BERNARD New Bloomfield, Pa.
V. M. D., University of Pennsylvania, 1918
Vouchers: Ray O. Whipple and T. E. Munce.
- SELF, RICHARD A. Route 8, Box 675, Dallas, Texas
D. V. M., A. & M. College of Texas, 1927
Vouchers: N. F. Williams and W. R. McCuiston.

- SEVERN, S. S. Seguin, Texas
D. V. S., Kansas City Veterinary College, 1911
Vouchers: N. F. Williams and W. R. McCuiston.
- SHAHAN, M. S. Box 338, Sacramento, Calif.
D. V. M., Colorado Agriculture College, 1924
Vouchers: W. L. Curtis and Joseph M. Arburua.
- SHAW, CORVIN VICTOR 7019 South Sangamon St., Chicago, Ill.
D. V. M., United States College of Veterinary Surgeons, 1915
Vouchers: Robert J. Robertson and H. Busman.
- SHEARER, HARPER HENRY 4444 Woodlawn Ave., Chicago, Ill.
D. V. M., Iowa State College, 1920
Vouchers: H. Busman and Wm. F. Moore.
- SKILES, JACK L. Box 652, Denton, Texas
D. V. M., Kansas City Veterinary College, 1916
Vouchers: N. F. Williams and W. R. McCuiston.
- SMITH, WILLIAM HENRY JR.
Bureau of Animal Industry, U. S. Dept. of Agr., Washington, D. C.
V. M. D., University of Pennsylvania, 1897
Vouchers: A. J. Pistor and A. E. Wight.
- STEERE, T. H. 324 South Pleasant St., Belding, Mich.
D. V. M., Grand Rapids Veterinary College, 1909
Vouchers: Wm. Hansen and B. J. Killham.
- SUTHERLAND, R. C. 2419 Market St., San Diego, Calif.
D. V. M., Terre Haute Veterinary College, 1912
Vouchers: A. C. Rosenberger and W. L. Curtis.
- TRUBEY, GEORGE WILLIAM Colon, Mich.
D. V. M., Michigan State College, 1919
Vouchers: Theodore S. Rich and M. P. Hunt.
- TUCKERMAN, EDWIN DOWNING 331 W. 2nd St., Media, Pa.
V. M. D., University of Pennsylvania, 1928
Vouchers: Ira Mitterling and W. S. Gimper.
- UMBERGER, GAIL M. Harveyville, Kans.
D. V. M., Kansas State Agricultural College, 1919
Vouchers: R. F. Coffey and C. B. Clement.
- WARD, RALEIGH M. Clayton, Ind.
D. V. M., Kansas City Veterinary College, 1912
Vouchers: C. A. Clawson and Benj. H. Yenner.
- WATT, JOHN WILLIAM 409 Savin Ave., West Haven, Conn.
B. V. Sc., Ontario Veterinary College, 1923
Vouchers: Edwin Laitinen and B. D. Radcliff.
- WEBER, RALPH E. Coleridge, Nebr.
D. V. M., Iowa State College, 1929
Vouchers: H. D. Bergman and W. F. Guard.
- WEST, LESLIE W. 4523 Emerald Ave., Chicago, Ill.
D. V. M., Indiana Veterinary College, 1923
Vouchers: H. Busman and L. Enos Day.
- WEST, PATRICK F. 7925 St. Lawrence Ave., Chicago, Ill.
V. M. D. University of Pennsylvania, 1913
Vouchers: Ira Mitterling and T. E. Munce.
- WHATLEY, DAVID EARL 2100 Lincoln St., Topeka, Kans.
D. V. M., Colorado Agricultural College, 1922
Vouchers: F. A. Imler and C. B. Clement.
- WHITE, FLOYD HENRY 812 3rd St., San Rafael, Calif.
D. V. M., Cornell University, 1920
Vouchers: Joseph M. Arburua and John McInnes.
- WHYTOCK, JOSEPH S. 3467 La Crescenta Blvd., Verdugo City, Calif.
D. V. M., State College of Washington, 1921
Vouchers: W. L. Curtis and J. P. Bushong.

- WILLIAMS, ARTHUR HENRY Williams, Iowa
D. V. M., St. Joseph Veterinary College, 1922
Vouchers: C. C. Donelson and C. L. Campbell.
- WINGATE, JAMES M. Waynesboro, Miss.
D. V. M., Alabama Polytechnic Institute, 1928
Vouchers: E. Heiny and Hartwell Robbins.
- YETTER, SIMEON 402½ Penn St., Huntingdon, Pa.
M. D. V., McKillip Veterinary College, 1909
Vouchers: H. Busman and Robert J. Robertson.

Applications Pending

SECOND LISTING

- Asmus, R. A., 100 Oakwood Ave., Troy, N. Y.
Atkins, Hazen S., 97 Perry St., Pontiac, Mich.
Audinwood, Harry George, Tioga, Pa.
Baer, Louis S., 1099 Elmwood Ave., Columbia, S. C.
Barger, Seth E., 409 N. B. Street, Ft. Smith, Ark.
Baver, Alvin Franklin, 248 E. Main St., Kutztown, Pa.
Beattie, Benjamin Myers, Chambersburg, Pa.
Berry, Hugh Elmer, R. F. D. No. 6, Staunton, Va.
Boman, Thomas W., Box 406, Laurel, Miss.
Boyd, Homer C., Lake Village, Ark.
Bradley, Alfred Martin, Alvord, Iowa.
Burk, James Arthur, Shippensburg, Pa.
Burnette, Clarence Arthur, 62 Torringford St., Winsted, Conn.
Buttfield, Lynnford C., 629 Sinclair St., Reno, Nev.
Caldwell, Capt. George L., Medical Field Service School, Carlisle Barracks, Pa.
Caldwell, John W., 527 Central Ave., Hemet, Calif.
Campbell, Larkin S., 1629 Fillmore, Topeka, Kans.
Card, Bowen Oliver, Sylvania, Pa.
Carr, Frank H., 722 State Office Bldg., Lansing, Mich.
Church, Dwight S., 626 Liberty St., Erie, Pa.
Coover, Eugene M., 147 West 18th St., Erie, Pa.
Corkill, Joseph J., 317 E. Patterson St., Lansford, Pa.
Davies, Montague A., Troy, Pa.
Davis, Charles Louis, 303 Livestock Exchange Bldg., Denver, Colo.
Defendorf, Horatio Ernest, Grand Blanc, Mich.
Denham, William Burton, Cumberland, Iowa.
Dickman, Andrew Jackson, 1120 N. 6th St., Boise, Idaho.
Denson, Leonidas L. Jr., Box 42, Ackerman, Miss.
Derk, Miles Raymond, 124 S. Broad St., Jersey Shore, Pa.
De Turck, Daniel H., Oley, Pa.
Dockstader, Hall Andrew, Rudd, Iowa.
Donovan, Lt. Col. Andrew E., c/o Adjutant General, U. S. Army, Washington, D. C.
Drake, Francis B., 3630 Edwards Road, Hyde Park, Cincinnati, Ohio.
Dunn, Ralph Alexander, 511 E. Water St., Charlottesville, Va.
Dykema, Herman, R. No. 4, Muskegon, Mich.
Ehlers, Daniel P., Box 403, Harrisburg, Pa.
Eliason, Theodore O., Kerkhoven, Minn.
Emery, Ashton, W., 97 Perry St., Pontiac, Mich.
Fenstermacher, James O., New Tripoli, Pa.
Fisher, William F., Box 155, Yerington, Nev.
Fitz Gerald, Gerald W., Army Vet. School, Army Med. Center, Washington, D. C.
Ford, John Arnold, 909 W. Anaheim St., Long Beach, Calif.
Foss, Ole D., Christine, N. Dak.
Gage, Fred B., 1822 N. Alexandria Ave., Los Angeles, Calif.
Garrahan, Lorenzo P., Florida 668, Buenos Aires, Argentina.
Gaw, John Howard, Wythesville, Va.

Genre, T. T., 722 State Office Bldg., Lansing, Mich.
Giffey, Joe W., Starkville, Miss
Gilbert, I. P., Courtland, Va.
Gildow, E. M., University of Idaho, Moscow, Idaho.
Greer, Lt. Charles S., Fort Bliss, El Paso, Texas
Haines, W. Albertson, Bristol, Pa.
Hannon, Joseph, 130 W. 6th St., Davenport, Iowa.
Harris, Frank Thomas, Box 852, Twin Falls, Idaho.
Henderson, Clarence B., c/o Leavenworth Pkg. & Stor. Co., Leavenworth, Kans.
Herrold, William C., 3129 Spring Grove Ave., Cincinnati, Ohio.
Hiatt, William L., 212 Live Stock Exch. Bldg., Stock Yds. Sta., Oklahoma City, Okla.
Hobdy, William Madison, Box 15, Salem, Va.
Hoffmire, Colenzo H., Franklin Alley, Adrian, Mich.
Horner, Ernest Allen, Brighton, Iowa.
Hovorka, Joseph W., 6213 W. 22nd St., Berwyn, Ill.
Hurd, Ray B., Box 202, Nampa, Idaho.
Johnson, E. E., 4529 Lodewyck, Detroit, Mich.
Keelor, Allen Z., Gratersford, Pa.
Kemp, Donald T., Dept. of Health, 1300 Beaubien St., Detroit, Mich.
Kingan, John C., 4082 Liberty Ave., Pittsburgh, Pa.
Klug, John Henry, Random Lake, Wis.
Lawton, Russell G., 310 State House, Providence, R. I.
Lewis, Capt. Ralph Henry, Fort Sam Houston, Texas.
Little, Harry Joseph, 515 Market St., Williamsport, Pa.
Lossie, Fred H. Harbor Creek, Pa.
Lubbehusen, Raymond Elmer, Box 403, Harrisburg, Pa.
Ludins, George H., 203 Affleck St., Hartford, Conn.
Lynch, Walter P., Towanda, Pa.
McCabe, James C., Box 211, Iowa City, Iowa.
McCoy, Ellis E., 408 Park St., Charlottesville, Pa.
Madsen, David E., Box 403, Harrisburg, Pa.
Michener, Hiram M., North Wales, Pa.
Miller, Frank G., 719 8th Ave., Lewiston, Idaho.
Mitchell, George Elmer, 3330 Eastside Ave., Cincinnati, Ohio.
Musselman, Eugene E., King and Queen Court House, Va.
Nissley, S. M., 16 Spring St., Bellefonte, Pa.
Owens, Lt. John Lloyd, Fort Sam Houston, Texas.
Pease, W. W., 448 North St., Meadville, Pa.
Pilgrim, Ralph V., Clarksville, Texas.
Pless, Louis R., 16630 Washburn Ave., Detroit, Mich.
Poseiner, Wm., 922 Nassau St., Cincinnati, Ohio.
Powell, Thomas Maurice, 27 East Markison Ave., Columbus, Ohio.
Ramsey, Capt. Mott, Fort Ringgold, Texas.
Rasmussen, George C., Kimballton, Iowa.
Ratts, Floyd S., 1024 Laramie, Manhattan, Kans.
Rea, Herbert E., West Branch, Mich.
Reading, Claude Harvey, Hopkins, Mich.
Reagor, Harry Abner, 30 California Ave., Reno, Nevada.
Reddert, Fred E., Independence, Calif.
Reed, Charles W., R. F. D. 4, Gibsonia, Pa.
Reichley, John Ralph, Dover, Pa.
Richey, Alfred N., Lanesboro, Iowa.
Rile, Edward A., Blue Bell, Pa.
Ritter, A. B., Pennsburg, Pa.
Rivers, Reuben N., Lansdale, Pa.
Rosekrans, William R., 1210 Evergreen Ave., Bronx, New York, N. Y.
Rouse, Palmer Ladd, 2203 Peach St., Erie, Pa.
Ruby, John Louis, 4036 Platt Ave., Fresno, Calif.
Rudolph, James Abram, 341 Terrace Ave., Cincinnati, Ohio.
Ruth, Vincent Moyer, Franconia, Pa.

Sherrick, George D., Smithfield Hotel, Smithfield, Pa.
 Shimer, W. S., Box 355, Altoona, Pa.
 Skelton, James A., Prairie Grove, Washington Co., Ark.
 Smith, Hilton A., State College of Washington, Pullman, Wash.
 Smith, Wilbur C., 219 E. 10th St., Waterloo, Iowa.
 Snyder, John C., Elderton, Pa.
 Stein, Clarence Dinsmore, Box 586, Pearl River, N. Y.
 Still, Seaborn Hassell, Box 393, Suffolk, Va.
 Strickler, Jacob Etter, Chambersburg, Pa.
 Sturrock, Alexander P., Waterford, Pa.
 Super, Daniel H. Warrensville, Pa.
 Taylor, Brainard Louis, Emmett, Idaho.
 Telford, Roswell A., 914 Reber St., Waterloo, Iowa.
 Tierney, Daniel David, 8055 S. Bishop St., Chicago, Ill.
 Underwood, Paul Clifford, 4409 Sidney Ave., Chicago, Ill.
 Unertl, John T., 380 4th St., Milwaukee, Wis.
 Upton, Clarence, c/o Evansville Packing Co., Evansville, Ind.
 Vanderloo, Vivian Bernard, 870 West 14 St., Dubuque, Iowa.
 Van Vlandren, Cornelius, 610 Rubber Ave., Naugatuck, Conn.
 Waddle, Harry Patton, 401 Walnut St., Hattiesburg, Miss.
 Wagner, C. Arthur, 1210 Park Ave., Williamsport, Pa.
 Walter, Harry K., 318 Livestock Exch., Herrs Island, Pittsburgh, Pa.
 Warwick, John G., 3442 S. 15th Ave., Seattle, Wash.
 Waters, Capt. Fred C., Presidio of Monterey, Calif.
 Webb, Edw. A., 513 N. Pennsylvania Ave., Avondale, Pa.
 Whyte, William Burnette, 9 Gerald Ave., Highland Park, Mich.
 Wight, Capt. Allen C., Fort Sam Houston, Texas.
 Woods, John W., Mendenhall, Miss.
 Young, John O., 626 Clay St., Topeka, Kans.

The amount which should accompany an application filed this month is \$7.50, which covers membership fee and dues to January 1, 1930, including subscription to the JOURNAL.

Motor to the motor metropolis, August 13-16.

Castrating and Docking Improves Lambs

Because of the fact that the largest part of the return from farm flocks is derived from the sale of lambs, it is highly desirable to have the lambs in the best possible condition for market. This fact is emphasized in Farmers' Bulletin No. 1134-F, "Castrating and Docking Lambs," recently published by the U. S. Department of Agriculture. Lambs which have been castrated and docked are better developed and are more uniform and attractive on the market than other lambs, according to specialists of the Department. Ram lambs and undocked lambs are discriminated against because they are less well developed and lack a uniform and attractive appearance. Lambs that have not been castrated do not produce the same gains nor do they show the same good condition as observed in those that have been castrated.

Copies of the bulletin may be obtained free by writing the U. S. Department of Agriculture, Washington, D. C.

COMING VETERINARY MEETINGS

- Alabama Veterinary Medical Association. Montgomery, Ala. July 1, 1929. Dr. C. A. Cary, Secretary, Alabama Polytechnic Institute, Auburn, Ala.
- San Diego-Imperial Veterinary Medical Association. San Diego, Calif. July 3, 1929. Dr. A. P. Immenschuh, Secretary, Santee, Calif.
- Manitoba, Veterinary Medical Association of. Brandon, Man. July 5, 1929. Dr. Wm. Hilton, Secretary, 510 Greenwood Place, Winnipeg, Man.
- Montana Veterinary Medical Association. Missoula, Mont. July 8-9, 1929. Dr. Hadleigh Marsh, Secretary, Helena, Mont.
- Missouri Valley Veterinary Association. Omaha, Nebr. July 8-10, 1929. Dr. E. R. Steel, Secretary, 8023 Wornall Rd., Kansas City, Mo.
- Chicago Veterinary Society. Great Northern Hotel, Chicago, Ill. July 9, 1929. Dr. J. B. Jaffray, Secretary, 2956 Washington Blvd., Chicago, Ill.
- Kansas City Association of Veterinarians. New Baltimore Hotel, Kansas City, Mo. July 9, 1929. Dr. J. D. Ray, Secretary, 400 New Centre Bldg., Kansas City, Mo.
- Eastern Iowa Veterinary Association. Riverside Park, Cedar Valley Country Club, Vinton, Iowa. July 9, 1929. Dr. R. M. Hofferd, Secretary, Box 544, Cedar Rapids, Iowa.
- Georgia State Veterinary Association. Henry Grady Hotel, Atlanta, Ga. July 9-10, 1929. Dr. Peter F. Bahnsen Secretary, Americus, Ga.
- Oklahoma State Veterinary Medical Association. Bartlesville, Okla. July 9-10, 1929. Dr. C. H. Fauks, Secretary, 1919 W. Ash St., Oklahoma City, Okla.
- Kentucky Veterinary Medical Association. Louisville, Ky. July 10-11, 1929. Dr. C. G. Kreidler, Secretary, Maysville, Ky.
- Minnesota State Veterinary Medical Association. University Farm, St. Paul, Minn. July 10-11, 1929. Dr. C. P. Fitch, Secretary, University Farm, St. Paul, Minn.
- Northwestern Ohio Veterinary Medical Association. Wallbridge Park, Toledo. July 11, 1929. Dr. F. A. Lambert, Secretary, c/o Columbus Serum Co., Sta. C, Box 53, Columbus, Ohio.

- North Dakota Veterinary Medical Association. Fargo, N. Dak. July 11-12, 1929. Dr. Lee M. Roderick, Secretary, State College Station, Fargo, N. Dak.
- New Jersey Veterinary Medical Association of. Asbury Park, N. J. July 11-12, 1929. Dr. E. R. Cushing, Secretary, Box 536, New Brunswick, N. J.
- Wisconsin Veterinary Medical Association. Wisconsin Rapids, Wis. July 15-16, 1929. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.
- British Columbia, Washington and Oregon Veterinary Medical Associations. Vancouver, B. C. July 15-17, 1929. Dr. Clifford Ackley, Secretary, Winlock, Wash.
- South Carolina Association of Veterinarians. Hartsville, S. C. July 16-17, 1929. Dr. M. R. Blackstock, Secretary, 157 Hampton Ave., Spartansburg, S. C.
- Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. July 17, 1929. Dr. W. L. Curtis, Secretary, 1264 W. 2nd St., Los Angeles, Calif.
- Nevada State Veterinary Association. Reno, Nevada. July 17, 1929. Dr. Edward Records, Secretary, University of Nevada, Reno, Nevada.
- Saskatchewan, Veterinary Association of. University of Saskatchewan, Saskatoon, Sask. July 17-19, 1929. Dr. R. G. Chasmar, Secretary, Hanley, Sask.
- Maryland State Veterinary Medical Association. Medical Hall, 1211 Cathedral St., Baltimore, Md. July 19-20, 1929. Dr. E. M. Pickens, Secretary, College Park, Md.
- Illinois State Veterinary Medical Association. Jefferson Hotel, Peoria, Ill. July 17-18, 1929. Dr. W. H. Welch, Secretary, Lexington, Ill.
- Virginia State Veterinary Medical Association. Hotel George Washington, Winchester, Va. July 25-26, 1929. Dr. George C. Faville, Secretary, Hampton, Va.
- Hudson Valley Veterinary Medical Society. Kenozia Lake, Ulster County, N. Y. August 6, 1929. Dr. J. G. Wills, Secretary, 122 State St., Albany, N. Y.
- Connecticut Veterinary Medical Association. Norwich, Conn. August 7, 1929. Dr. E. H. Patchen, Secretary, 83 New Haven Ave., Milford, Conn.
- American Veterinary Medical Association. Book-Cadillac Hotel, Detroit, Mich. August 13-16, 1929. Dr. H. Preston Hoskins, Secretary, 716 Book Bldg., Detroit, Mich.

THE ANTIGENIC VALUE OF FORMOLIZED BOTULINUM TOXINS*

By ROBERT GRAHAM, E. A. TUNNICLIFF, E. C. McCULLOCH
and FRANK THORP, JR.

*Laboratory of Animal Pathology and Hygiene,
University of Illinois, Urbana, Ill.*

The hope of obtaining an active immunizing agent for botulism in animals comparable to formolized diphtheria toxin, as employed in immunizing man, prompted experiments to determine: first, if formalin would detoxify botulinum toxins A, B and C and, second, if botulinum toxins altered by formalin possessed antigenic properties. Preliminary results with B and C formolized botulinum toxins suggested that they could be detoxified with formalin and heat without altering their antigenic value. These findings, presented elsewhere,¹ suggested the advisability of attempting to detoxify type A toxin and to determine if the altered atoxic toxin was antigenic. It is the purpose of this paper to report experiments corroborating the preliminary observations on B and C toxoid and also on the effect of formalin and heat on type A toxin. In these studies it has been possible to make limited observations on the stability of botulinum toxoids and on the value of single and combined toxoids simultaneously injected into animals experimentally. Although the antigenic value of certain formolized botulinum toxins is clearly suggested, work has yet to be done on methods of detoxifying to obtain the maximum antigenic power consistently. Likewise, the possible immunizing value of this antigen under field conditions remains to be established.

THE USE OF FORMALIN AS A DETOXIFYING AGENT

For many years the use of chemicals in altering or detoxifying bacterial exotoxins and endotoxins in the preparation of antigens has engaged the attention of immunologists. More than a quarter of a century ago, Salkowski (1898)² recognized the detoxifying effect of formalin on tetanus toxin. Similar observations were made by Lowenstein (1904)³ and others, though the use of formalin-detoxified bacterial exotoxins as immunizing agents was not definitely proposed until 1921, when Glenny and his co-

*Presented at the 65th annual meeting of the American Veterinary Medical Association, August 7-10, 1928, Minneapolis, Minnesota.

workers (1923),⁴ in two separate publications, suggested the possible use of formolized diphtheria toxin, or diphtheria toxoid, as a prophylactic agent in man. In 1923 and 1924, Ramon⁵ described an atoxic diphtheria antigen prepared by heating formolized diphtheria toxin. This antigen was called diphtheria anatoxin. The antigenic properties of diphtheria anatoxin described by Ramon have been corroborated by many different European investigators,^{6,7,8,9} as well as by Moloney (1926)¹⁰ and Fitzgerald (1927),¹¹ of the School of Hygiene, Toronto University.

The advantages of diphtheria anatoxin in the hyperimmunization of horses, as well as the value of the flocculation test in determining the potency of diphtheria anatoxin, as described by Ramon, were only partly confirmed by Feierabend (1926),¹² while the results of Moloney (1926) and Weld (1925)¹³ suggest that the flocculating test is but a relative guide in establishing potency. Following the experimental evidence of the antigenic value of formolized diphtheria toxin, public health officials in European countries and in Canada have recommended its use in the prophylaxis of diphtheria in man.

FORMOLIZED BOTULINUM TOXINS

Other bacterial exotoxins, including tetanus and botulinum as well as different bacterial endotoxins, filtrable viruses, and snake venoms, according to reports of different investigators, have been rendered relatively avirulent and antigenic by the addition of formalin. Weinberg and Goy (1924)^{14,15} first immunized rabbits by injecting formalin-detoxified botulinum toxin. By repeated injections of the detoxified toxin into rabbits they obtained a potent antitoxin. Their results clearly suggested the possibility of immunizing horses and other susceptible animals. Bronfenbrenner and Reichert (1926),¹⁶ in studying the toxin-antitoxin flocculating phenomenon of Ramon, also demonstrated that rabbits could be immunized with formolized botulinum toxin, while Buckley (1927-28),¹⁷ of the U. S. Bureau of Animal Industry, successfully immunized mules by repeated injections of formolized botulinum toxin. At about the same time, Theiler (1927)¹⁸ reported that Lugol's solution detoxified the parobotulinum or lamziekte (*Cl. botulinum* C) toxin for goats, but failed to note favorable results from other chemicals employed.

At the Illinois Experiment Station the results of preliminary experiments clearly demonstrated the antigenic value of formolized botulinum toxins B and C in guinea pigs and horses. Ani-

imals receiving proper amounts of potent toxoid withstood lethal doses of the homologous unaltered toxin ten days after a single injection of toxoid. While the results were not always consistent, they were sufficiently encouraging to prompt the treatment of healthy animals in clinical outbreaks of the disease and to stimulate an effort to detoxify botulinum toxin type A. Of the field results obtained nothing definite can be said other than to note that, with few exceptions, the experimental botulinum toxoids A, B and C did not exert a noticeable unfavorable effect following subcutaneous injection. In one instance, six of seventeen horses injected with appropriate amounts of A, B and C botulinum toxoids showed a transitory local reaction characterized by a swelling at the point of injection. The swellings disappeared in seven to ten days. Proof of immunization of animals under field conditions with formolized botulinum toxins was not obtained in our field observations, though the antigenic value of formolized toxins A, B and C was repeatedly observed at the laboratory in guinea pigs, rabbits, chickens, calves, horses and mules. Failure of the toxoid to protect some guinea pigs, chickens and horses also was noted. Though these unsuccessful cases were in the minority, the irregularities suggested the importance of non-lethal doses of toxoid and the possibility of other factors which might enter into the immunization complex, including individual peculiarities of the animal.

DETOXIFICATION OF BOTULINUM TOXINS A, B AND C WITH FORMALIN AND HEAT

The initial experiments with formolized botulinum toxins A, B and C were designed to obtain information regarding the value of formalin and heat as detoxifying agents. For this purpose varying amounts of formalin (.3 to .9 per cent) were added to filtered and unfiltered botulinum cultures. The formolized cultures and culture filtrates, after being heated, were tested at intervals for viability by seeding in culture media favorable for the growth of *Cl. botulinum*, while toxicity or atoxicity was determined by injecting subcutaneously an amount equivalent to 10,000 to 30,000 detoxified lethal doses of the heated formolized filtered and unfiltered cultures in guinea pigs weighing 300 to 325 grams.* The formalin-treated filtered and unfiltered cultures became non-viable and non-toxic after varying periods of time. Formolized sterile toxin has been tentatively regarded as relatively

*Guinea pigs weighing not less than 300 grams or more than 325 grams were preferred, but not always available.

non-toxic if guinea pigs remained healthy and maintained their weight following subcutaneous injection of 10,000 detoxified lethal doses of toxin. It was regarded as completely detoxified if three times this amount proved innocuous. Completely detoxified botulinum toxins protected guinea pigs weighing 300 grams, as did smaller non-lethal amounts of partially detoxified toxins. According to the above standards of testing toxicity in guinea pigs, the completely detoxified toxins have proven quite incapable of producing the disease in chickens, horses, or mules, in amounts that proved sufficient to protect against lethal doses of the toxin.

The rate of detoxification, as well as the degree of detoxification, apparently depends upon different factors. Some of these factors are not understood, but the degree of toxicity of the original toxin seems to be important. Under the same conditions as to formalin content and heat, a toxin containing 2000 g.p.l.d. per cubic centimeter was detoxified more rapidly than one containing 10,000 or more g.p.l.d. per cubic centimeter. Temperatures above 37°C., together with an increase in the amount of formalin, appear to hasten detoxification. Evidence of detoxification in formolized (.5 per cent formalin), botulinum toxins containing 4000 to 10,000 g.p.l.d. per cubic centimeter, was detected after seven days at 37° C., yet 30 to 60 days, or a longer period of time, at this temperature, was frequently necessary to eliminate all trace of toxicity in toxoids prepared from toxin containing 10,000 to 80,000 g.p.l.d. per cubic centimeter.

In testing the toxicity of different toxoids in guinea pigs it has been observed that a given amount of toxoid may prove harmless in one guinea pig or one group of guinea pigs of 300 grams weight, while to other guinea pigs of slightly less weight the same amount of toxoid may be fatal. To avoid the possibility of error in evaluating detoxification, three out of four guinea pigs (300 to 325 grams) used in the test should survive the subcutaneous injection of 30,000 detoxified g.p.l.d. of toxin. This precaution appears to be a fairly reliable safeguard in preventing the vaccination form of the disease in large animals.

TESTS OF THE ANTIGENIC PROPERTIES OF BOTULINUM TOXOIDS A, B AND C

Guinea pigs inoculated with formolized botulinum toxoids A, B and C that remained healthy were injected subcutaneously with 5 to 10 g.p.l.d. of the unaltered toxin to test the antigenic

value of the toxoid. Results obtained with type B are given in table I. In testing the antigenic value of botulinum toxoids, an arbitrary unit of toxoid was employed. This tentative unit was taken to represent 10,000 g.p.l.d. of detoxified toxin, if it protects guinea pigs weighing 300 grams against 5 to 10 g.p.l.d. of unaltered toxin, while 3 such units, or 30,000 completely detoxified g.p.l.d., must not produce ill effects in guinea pigs of the same weight (table I).

TABLE I—Antigenic character of botulinum toxoid B (guinea pigs)

GUINEA PIG	WEIGHT (GRAMS)	TREATMENT		RESULT
		TOXOID 12516B SUB- CUTANEOUSLY (6-9-27)	TOXIN B SUB- CUTANEOUSLY (6-19-27)	
12873	340	.008 unit*	8 g.p.l.d.	Died 6-22-27
12874	170	.08 unit	8 g.p.l.d.	Died 6-21-27
12875	210	.16 unit	8 g.p.l.d.	Died 6-20-27
12876	190	.32 unit	8 g.p.l.d.	Released healthy 6-29-27
12877	250	.64 unit	8 g.p.l.d.	Died 6-22-27
12878	310	.8 unit	8 g.p.l.d.	Released healthy 6-29-27
12879	320	1.6 units	8 g.p.l.d.	Released healthy 6-29-27
12880	260	Controls	8 g.p.l.d.	Died 6-21-27
12881	260		8 g.p.l.d.	Died 6-21-27

*The arbitrary unit of toxoid employed in this discussion represents 10,000 detoxified guinea pig lethal doses of toxin, provided it protects a 300-gram guinea pig against 5 to 10 lethal doses of toxin.

Botulinum toxoids of known antigenic value injected into guinea pigs required a minimum of ten days following injection before protection against the unaltered toxin could be demonstrated. Guinea pigs inoculated with known protective toxoids, that were exposed on the sixth, seventh and eighth days, invariably succumbed earlier than the controls or untreated pigs, suggesting that formolized botulinum antigen possesses an aggressive or a sensitizing effect. This negative phase in inoculated pigs is followed by a measurable resistance to lethal amounts of the unaltered toxin. It is possible that some guinea pigs that succumb following exposure on the tenth day may represent an individual failure of the guinea pig to elaborate antibodies rather than the absence of antigenic properties in the formolized toxin. An interval of more than ten days following the injection of the toxoid before exposure to unaltered toxin might therefore be advantageous in establishing the antigenic value of toxoids.

The injection of toxoids containing .9 per cent formalin yielded some evidence to show that this amount of formalin may reduce or destroy the antigenic properties of the toxoid prepared from toxins of low potency and that it may affect toxins of high potency after a given period of time. Toxoids containing .9 per cent formalin also showed a tendency to produce sloughing at the point of injection. Heating formolized toxoids for 7 to 14 days above 42° C. also seems to injure the antigenic properties. The proper amount of formalin to employ in the preparation of toxoids of maximum potency has not been determined, though .5 to .6 per cent formalin, plus heating at 37 to 42° C. for periods varying from one to four or more weeks, has given favorable results.

Botulinum toxoid A: No difficulty was experienced in detoxifying type A toxin, yet in the first attempts irregular results were encountered, in demonstrating its antigenic value. Subsequent formalin-treated type A toxins heated at 37° C. have proved antigenic as demonstrated in chickens and guinea pigs. (table II).

TABLE II—Antigenic character of botulinum toxoid A (chickens)

CHICKEN	WEIGHT (GRAMS)	TREATMENT		RESULT
		TOXOID 510A SUB- CUTANEOUSLY (8-6-27)	TOXIN A SUB- CUTANEOUSLY (8-18-27)	
1046	830	3 units	10 g.p.l.d.	Released healthy 8-24-27
1047	840	3 units	10 g.p.l.d.	Released healthy 8-24-27
1048	900	4 units	10 g.p.l.d.	Released healthy 8-24-27
1049	960	4 units	10 g.p.l.d.	Released healthy 8-24-27
1050	780	Controls	10 g.p.l.d.	Died 8-19-27
1051	770		10 g.p.l.d.	Died 8-19-27

Guinea pigs weighing 250 to 330 grams were given a subcutaneous injection of one-half to two units of type A toxoid 510. Ten days later, these animals were completely protected against 8 g.p.l.d. of type A toxin, while the control pig became moribund and died. Chickens varying in weight from 830 to 960 grams were given three and four units of type A toxoid 510. They were protected twelve days later against 10 g.p.l.d. of unaltered toxin, while control chickens, receiving the same amount of toxin, promptly died (table III). Type A botulinum toxoids, when administered in single doses of 20 to 30 units, did not protect horses weighing from 600 to 1100 pounds. The same difficulty was experienced in protecting horses against unaltered B and C

TABLE III—*Antigenic character of botulinum toxoid A (guinea pigs)*

GUINEA PIG	WEIGHT (GRAMS)	TREATMENT		RESULTS
		TOXOID 510A SUB- CUTANEOUSLY (7-18-27)	TOXIN A SUB- CUTANEOUSLY (7-28-27)	
568	250	.5 unit	8 g.p.l.d.	Released healthy 8-15-27
569	300	1 unit	8 g.p.l.d.	Released healthy 8-15-27
570	320	1.5 units	8 g.p.l.d.	Released healthy 8-15-27
571	330	2 units	8 g.p.l.d.	Released healthy 8-15-27
572	325	Control	8 g.p.l.d.	Sick 7-30-27. Moribund for 4 days. Destroyed

botulinum toxins, with single injections of type B and C toxoids. A second injection of A, B, or C botulinum toxoid in horses, one week following the first injection, completely protected against 10,000 g.p.l.d. of toxin subcutaneously administered seven days later. The results of two separate injections of type A toxoid (8489 and 5560), followed by exposure to lethal amounts of type A toxin, are shown in table IV.

TABLE IV—*Antigenic character of botulinum toxoid A (horses)*

HORSE	WEIGHT (LBS.)	TREATMENT			RESULT
		TOXOID SUB- CUTANEOUSLY (4-14-28)	TOXOID SUB- CUTANEOUSLY (4-21-28)	TOXIN A SUB- CUTANEOUSLY (4-28-28)	
Gray	1100	20 units 8489A	20 units 8489A	10,000 g.p.l.d.	Released healthy 5-17-28
B. C. Pony	700	20 units 5560A	20 units 5560A	10,000 g.p.l.d.	Released healthy 5-17-28
Bay	900	Control	Control	10,000 g.p.l.d.	Died 5-2-28

TABLE V—*Antigenic character of botulinum toxoid B (guinea pigs)*

GUINEA PIG	WEIGHT (GRAMS)	TREATMENT		RESULT
		TOXOID 2700B SUB- CUTANEOUSLY (11-21-27)	TOXIN B SUB- CUTANEOUSLY (12-1-27)	
7758	230	2 units	10 g.p.l.d.	Died 12-2-27
7759	300	3 units	10 g.p.l.d.	Released healthy 12-14-27
5760	200	Control	10 g.p.l.d.	Died 12-2-27

Botulinum toxoid B: Botulinum toxoid type B, prepared by adding formalin (.3 to .6 per cent) to type B toxin and heating to 37°C., protected experiment animals, as shown in tables V, VI and VII.

TABLE VI—Antigenic character of botulinum toxoid B (horses)

HORSE	WEIGHT (LBS.)	TREATMENT		RESULT
		TOXOID 1413B SUB- CUTANEOUSLY (9-2-27)	TOXIN B SUB- CUTANEOUSLY (9-12-27)	
Black	1050	20 units	10,000 g.p.l.d.	Died 9-17-27
Cream	950	30 units	10,000 g.p.l.d.	Died 9-15-27
Mule No brand	750	Control	10,000 g.p.l.d.	Died 9-17-27

TABLE VII—Antigenic character of botulinum toxoid B (horses)

HORSE	WEIGHT (LBS.)	TREATMENT			RESULT
		TOXOID SUB- CUTANEOUSLY (9-18-27)	TOXOID SUB- CUTANEOUSLY (9-24-27)	TOXIN B SUB- CUTANEOUSLY (10-1-27)	
L	850	20 units 1413B	20 units 1413B	10,000 g.p.l.d.	Released healthy 10-6-27
N	850	20 units 1267B	20 units 1267B	10,000 g.p.l.d.	Released healthy 10-6-27
F	1000	10 units 1794B	10 units 1794B	10,000 g.p.l.d.	Died 10-2-27
SM	1300	Control	Control	10,000 g.p.l.d.	Died 10-3-27

In table V an irregularity is noted, such as frequently occurred in pigs below 300 grams in weight. Two units failed to protect a 230-gram guinea pig, while 3 units were effective in a 300-gram guinea pig, suggesting that pigs under 300 grams are of less value or even undesirable for measuring the antigenic value of this toxoid. In table VI, the non-protective character of a single 20 to 30-unit dose of type B toxoid (1413) is illustrated in horses, while the same toxoid (1413B), as well as 1267B, provided complete protection in two separate doses at intervals of one week (table VII). Toxoid 1794B, given in ten-unit doses a week apart, failed to protect animals against the unaltered homologous toxin.

Botulinum toxoid C: The results of detoxifying botulinum toxoid type C are a repetition of those obtained with types A and B, though the difficulty in maintaining highly toxic C strains of *Cl. botulinum* has necessitated the use of larger doses in protecting animals against unaltered toxin.

TESTS OF THE ANTIGENIC VALUE OF BOTULINUM TOXOIDS A, B AND C COMBINED

The antigenic value of A, B and C botulinum toxoids, as established separately in susceptible animals experimentally, suggested the possibility of combining the three toxoids in a single simultaneous injection to induce protection against the three homologous toxins. Toxoids A, B and C, of known protective value, were mixed and injected subcutaneously into guinea pigs and horses in one, two and three doses, at intervals of one week, followed by exposure to the three unaltered toxins administered singly and simultaneously, seven to ten days later. Guinea pigs treated in this manner withstood a single injection of lethal amounts of one of the botulinum toxins, indicating that a single injection of combined A, B and C toxoids may protect against a single toxin ten days later. Two simultaneous injections of A, B and C toxoids a week apart provided similar protection but

TABLE VIII—*Antigenic character of combined botulinum toxoids A, B, and C* (guinea pigs)*

GUINEA PIG	WEIGHT (GRAMS)	TREATMENT			RESULT	
		TOXOID SUB- CUTANEOUSLY (5-19-28)	TOXOID SUB- CUTANEOUSLY (5-26-28)	TOXIN SUB- CUTANEOUSLY (6-2-28)		
1562	250	1 unit A 1 unit B 1 unit C	1 unit A 1 unit B 1 unit C	5 g.p.l.d. A	Released 6-12-28	
1571	260	1 unit A 1 unit B 1 unit C	1 unit A 1 unit B 1 unit C	2 g.p.l.d. A 2 g.p.l.d. B	Released 6-12-28	
1484	330	1 unit A 1 unit B 1 unit C	1 unit A 1 unit B 1 unit C	2 g.p.l.d. A 2 g.p.l.d. B 2 g.p.l.d. C	Died 6-5-28	
1477	270	Controls			5 g.p.l.d. A	Died 6-4-28
1559	240				2 g.p.l.d. A 2 g.p.l.d. B	Died 6-3-28
1480	230				2 g.p.l.d. A 2 g.p.l.d. B	Died 6-4-28
					2 g.p.l.d. C	

*Botulinum toxoids 1793A (ice-box temperature 11 months), 1267B (ice-box temperature 7 months), 1774C (ice-box temperature 8 months).

did not protect against A, B and C toxins combined. Occasionally guinea pigs survived lethal amounts of two of the homologous toxins, but seldom withstood the three toxins (tables VIII and IX).

TABLE IX—Antigenic character of combined botulinum toxoids A, B, and C* (guinea pigs)

GUINEA PIG	WEIGHT GRAMS	TREATMENT				RESULT
		TOXOID SUBCUTANEOUSLY (5-28-28)	TOXOID SUBCUTANEOUSLY (6-4-28)	TOXOID SUBCUTANEOUSLY (6-7-28)	TOXIN SUBCUTANEOUSLY (6-28-28)	
910	310	1 unit A 1 unit B 1 unit C	→	→	5 g.p.l.d. A 5 g.p.l.d. B 5 g.p.l.d. C	Died 6-30-28
443	315	1 unit A 1 unit B 1 unit C	→	→	5 g.p.l.d. A 5 g.p.l.d. B 5 g.p.l.d. C	Released healthy 7-8-28
1492	330	1 unit A 1 unit B 1 unit C	1 unit A 1 unit B 1 unit C	1 unit A 1 unit B 1 unit C	5 g.p.l.d. A 5 g.p.l.d. B 5 g.p.l.d. C	Died 7-2-28
1560	310	Control	Control	Control	5 g.p.l.d. A 5 g.p.l.d. B 5 g.p.l.d. C	Died 6-30-28

*Botulinum toxoids 1793A, 1267B, 1774C.

TABLE X—Antigenic character of combined botulinum toxoids A, B and C* (horses)

HORSE	TREATMENT				RESULT
	TOXOID SUBCUTANEOUSLY (4-23-28)	TOXOID SUBCUTANEOUSLY (4-30-28)	TOXOID SUBCUTANEOUSLY (5-6-28)	TOXIN SUBCUTANEOUSLY (5-13-28)	
L (1100 lbs.)	30 units A 30 units B 12 units C 72 units	30 units A 30 units B 12 units C 72 units	→	→	Died 5-6-28
X (700 lbs.)	30 units A 30 units B 60 units	30 units A 30 units B 60 units	→	5000 A—g.p.l.d. 5000 B—g.p.l.d.	Died 5-17-28
A (1200 lbs.)	30 units A 30 units B 12 units C 72 units	20 units A 20 units B 8 units C 48 units	20 units A 20 units B 8 units C 48 units	5000 A—g.p.l.d. 5000 B—g.p.l.d.	Released healthy 5-23-28
E (800 lbs.)	Control	Control	Control	5000 A—g.p.l.d. 5000 B—g.p.l.d.	Died 5-14-28

*Botulinum toxoids 8489A, 1413B, and 1674C. (Toxoids 1413B and 1674C at ice box temperature 7 months).

TABLE XI—The effect of room temperature on botulinum toxin A (guinea pigs)

GUINEA PIG	WEIGHT (GRAMS)	TOXOID	DETOXIFICATION		ROOM TEMPERATURE	TREATMENT					RESULTS
			FORMALIN	HEAT		Toxoid 1st Day	Toxoid 7th Day	Toxin 10th Day	Toxoid 14th Day	Toxin 21st Day	
740 379	320 370	510A 510A	.5 per cent	26 da.-37°C.	12 months	1 unit 2 units	1 unit ↑	↑ 10 g.p.l.d.	3 units	10 g.p.l.d.	Remained healthy Died—24 hours
746 846	350 370	1277A 1277A	.75 per cent	6 da.-37°C.	11 months	1 unit 2 units	1 unit ↑	↑ 10 g.p.l.d.	3 units	10 g.p.l.d.	Remained healthy Died—48 hours
914 303	490 330	5733A 5733A	.75 per cent	7 da.-37°C.	6 months	1 unit 2 units	1 unit ↑	↑ 10 g.p.l.d.	3 units	10 g.p.l.d.	Died—72 hours Died—48 hours
841 909 313 342	300 320 330 300	2704A 2704A 2702A 2702A	.75 per cent	7 da.-37°C.	7 months	1 unit 2 units 1 unit 2 units	1 unit ↑ 1 unit ↑	↑ 10 g.p.l.d. ↑ 10 g.p.l.d.	3 units 3 units	10 g.p.l.d. 10 g.p.l.d.	Remained healthy Died—48 hours Remained healthy Died—48 hours
745 748	320 320		Controls			↑ ↑	↑ ↑	↑ 10 g.p.l.d.	↑	10 g.p.l.d.	Died—20 hours Died—20 hours

TABLE XII—*The effect of room temperature on botulinum toxoid B (guinea pigs)*

GUINEA PIG	WEIGHT (GRAMS)	TOXOID	DETOXIFICATION		ROOM TEMPERATURE	TREATMENT					RESULTS
			FORMALIN	HEAT		TOXOID 1ST DAY	TOXOID 7TH DAY	TOXIN 10TH DAY	TOXOID 14TH DAY	TOXIN 21ST DAY	
744	250	1413B	.5 per cent	6 da.—37°C.	7 months	1 unit	1 unit	—	3 units	5 g.p.l.d.	Remained healthy
912	240	1413B				2 units	—	5 g.p.l.d.	—	—	Died—24 hours
919	280	2700B		3 da.—37°C.	5 months	1 unit	1 unit	—	3 units	5 g.p.l.d.	Remained healthy
339	220	2700B				2 units	—	5 g.p.l.d.	—	—	Died—24 hours
438	240	2703B	Controls			1 unit	1 unit	—	3 units	5 g.p.l.d.	Remained healthy
329	170	2703B				2 units	—	5 g.p.l.d.	—	—	Died—24 hours
901	260					—	—	—	—	—	Died—24 hours
847	240					—	—	—	—	—	Died—24 hours

TABLE XIII—The effect of room temperature on *Botulinum Toxoid C* (guinea pigs)

GUINEA PIG	WEIGHT (GRAMS)	TOXOID	DETOXIFICATION		ROOM TEM- PERATURE	TREATMENT					RESULTS
			FORMALIN	HEAT		TOXOID 1st Day	TOXOID 7th Day	TOXIN 10th Day	TOXOID 14th Day	TOXIN 21st Day	
835 432	240 290	1674C 1674C	.75 per cent	42°C.-48 hr. 37°C.-4 da.	8 months	1 unit 2 units	1 unit —	— 5 g.p.l.d.	3 units	4 g.p.l.d.	Remained healthy Died—48 hours
431 325	320 280	1774C 1774C	.75 per cent	37°C.-4 da.	8 months	1 unit 2 units	1 unit —	— 5 g.p.l.d.	3 units		Died—24 hours Died—24 hours
322 708	250 270	1787C 1787C	.75 per cent	42°C.-24 hr.	7 months	1 unit 2 units	1 unit —	— 5 g.p.l.d.			Died Died—48 hours
742 440	270 220	2813C 2813C	.75 per cent	37°C.-7 da.	6 months	1 unit 2 units	1 unit —	— 5 g.p.l.d.	3 units	4 g.p.l.d.	Remained healthy Lost
337 922	250 230		Controls			— —	— —	— 5 g.p.l.d.	—	4 g.p.l.d.	Died—48 hours Died—24 hours

In attempting to protect horses against A, B and C unaltered toxins, maximum doses of the combined toxoids were injected subcutaneously at weekly intervals (table X). Horse L, weight 1100 pounds, was given 30 units of type A toxoid, 30 units of type B toxoid, and 12 units of type C toxoid simultaneously at intervals of seven days. This amount of toxoid represents 1,440,000 detoxified g.p.l.d. Death occurred suddenly, seven days after the second injection. Comparable amounts were administered to horse X, weight 700 pounds. This animal died four days after receiving 5000 g.p.l.d. of A toxin, and 5000 g.p.l.d. of B toxin subcutaneously. Horse A, weight 1200 pounds, given 30 units of A, and 30 units of B, followed by two smaller doses at weekly intervals, survived both A and B unaltered toxins.

STABILITY OF BOTULINUM TOXOIDS A, B AND C

A series of botulinum toxoids A, B and C, that were detoxified with formalin and incubated five to seven days at 37 to 42°C., were injected into guinea pigs to establish their non-lethal as well as their antigenic properties. Each toxoid, given in 1 to 2-unit doses, protected guinea pigs against 5 to 10 g.p.l.d. of homologous toxin. The protective toxoids were then placed at room temperature and after 5 to 12 months tested for potency (tables XI, XII and XIII). It was shown that all toxoids, kept at room temperature from 5 to 7 months, depreciated in antigenic value. Following exposure at room temperature, two units consistently failed to protect, while two doses of one unit each plus one injection of three units at seven-day intervals, protected guinea pigs against ten lethal doses of toxin. A separate dose of five units of the weakened toxoid kept at room temperature for several months seemed equivalent to one unit of A, one unit of B, and one unit of C of the freshly prepared antigen, as measured in guinea pigs. Since these toxoids when freshly prepared protected guinea pigs in single doses of one to two units, it seems apparent that room temperature may lower the antigenic value of formalized botulinum toxoids. Toxoids kept at ice-box temperature have remained apparently unaltered in potency for more than one year.

SUMMARY

A summary of the results of preliminary experiments suggests that formalin and heat may detoxify botulinum toxins without altering a measurable amount of antigenic substance. Amounts of detoxified toxins necessary to protect guinea pigs, chickens and

horses against lethal amounts of the toxin consistently failed to produce the disease and, with certain exceptions, protected animals against lethal amounts of homologous toxin. The preliminary findings seem to justify the hope that this antigen ultimately may be perfected and employed advantageously in emergency to immunize animals under field conditions.

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DISCUSSION

DR. C. E. SALSBERY: Undoubtedly practitioners who are in territories where botulism is not prevalent will not be as much interested in this subject as those in territories where the disease is encountered regularly. There are certain sections of the country where immunization and treatment of botulism constitute a more or less prominent part of the veterinarian's work.

Up to this time, the only means of combatting the disease has been through the use of botulinus antitoxin. It has been expensive treatment and when used as an immunizing agent has not been altogether satisfactory because only temporary immunity has been obtained. When used as treatment in affected animals it is effective only when given early and in fairly large amounts. This is especially true in treating horses and cattle. In treating chickens affected with botulism or "limberneck" the results are more satisfactory and in these cases it has been much more effective as a cure.

Botulism occurs in three distinct types, namely, A, B and C. The antitoxin of one type has no effect whatever against the other two. Because of this condition it is necessary to prepare a polyvalent serum containing antitoxins for all three types. In order to prepare an effective mixture, each of the antitoxins must be of high unitage, because when they are mixed it can be seen that each will be diluted. Thus, if type A antitoxin has a unitage of 300 per cubic centimeter before mixing, each cubic centimeter of the mixture will have considerably less than 300 units of type A. The same rule applies to the other types as well. The successful hyperimmunization of horses and cattle is exceedingly difficult and requires a long period of injections, and a great many animals cannot be made to show any degree of unitage in comparison with what is necessary to make good antitoxin. That is what makes the antitoxin expensive, and a new product, less expensive and one that produces active immunity, should be welcomed.

The discovery of toxoids or anatoxins made according to the method discussed in the paper read by Dr. Graham opened up a very practical field of active immunization, not alone against botulism but against tetanus and

diphtheria as well. The latter is already in use as an immunizing agent against diphtheria, and the results are reported as being highly satisfactory.

Experimental data in connection with botulinus toxoid or anatoxin show it to be a product of exceptional merit. The possibility of the production of an active immunity is already an accomplished fact. We have been working with it for nearly two years and have been successful in immunizing guinea pigs, rabbits, chickens, cattle and horses. The results of some of the work have already been published.

There are yet some questions that must be answered regarding the stability of the product, the permanency of the immunity and proper field dosage. Our present knowledge indicates that the toxoid or anatoxin is quite stable, at least the records do not show any serious deterioration up to six months. The reports of other investigators indicate that a fairly stable and lasting immunity is produced in animals vaccinated with it, and also that it is possible to use the toxoid alone in hyperimmunizing horses for the production of antitoxin. That, at least, would indicate the reliability of the anatoxin as an active antigen.

It is much easier to fix a satisfactory dose for experimental purposes than it is to fix one for general field use. We feel certain that the field dosage for chickens has been determined and probably also for cattle. There is such a great difference in the degree of susceptibility between these animals and horses that there has been considerable difficulty experienced in arriving at the proper universal dose for this animal. A single injection appears to be sufficient for chickens and cattle, but horses require more. We believe that a double method of vaccination for horses will securely immunize them; at least our data at this time suggest that as the most suitable method for this animal.

Undoubtedly there is sufficient evidence at hand to support the belief that botulinus anatoxin will eventually be used as a very successful means of protecting farm animals, at least chickens, cattle and horses, against botulism.

DR. H. C. H. KERNKAMP: When I was on my feet before, I confined myself to hog cholera virus entirely, but I might mention that Dr. Kramer, the inventor of the electro-positively charged filter, says that botulinus toxin was innocuous after passage through one of these filters. Likewise, that the fraction of diphtheria toxin responsible for acute death is also retained by the filter.

DR. F. C. KOCHENDORFER: What is the average dose for horses in case of botulism on infected premises, where there are seven or eight horses on the place and you were to immunize them?

DR. GRAHAM: We have done that experimentally and have used from 15 to 30 units, but I could not recommend any particular dose, because I feel it is a point where we should know exact dosage. In outbreaks, all the horses have been given 15 to 30 units, depending on the size of the horses, and repeated in seven days, and seven days later we have suggested that they put the animals back on feed that was suspected. In a general way, from 15 to 30 units, and withhold them from feed about seven days after the second injection; that is the plan I have been using.

DR. KOCHENDORFER: I have used 40 to 60 cc and I would repeat it in from three to five days. I have tried it in seven to ten different herds and have had poor results. Probably out of 150 horses, we lost forty, worth \$175 to 250 apiece.

DR. GRAHAM: The amount of toxin is always an item. We could feed, in silage, overpowering doses of toxin that antitoxin would not protect against and in carrying out these toxoid tests we must endeavor to protect the animals against overdosage.

DR. KOCHENDORFER: The horses were on various farms, some just out of the pasture, others in the barn. Some got ensilage, some oats and hay, others corn and straw, etc. We tried everything possible.

DR. GRAHAM: If I had that experience I would try to check my diagnosis by holding an autopsy. Take material to the laboratory and be sure of the diagnosis, because there might be something else involved there. At least it is worth looking into.

DR. KOCHENDORFER: We held postmortems but that did not help.

DR. GRAHAM: The organism should be isolated. I believe you are having something out of the ordinary.

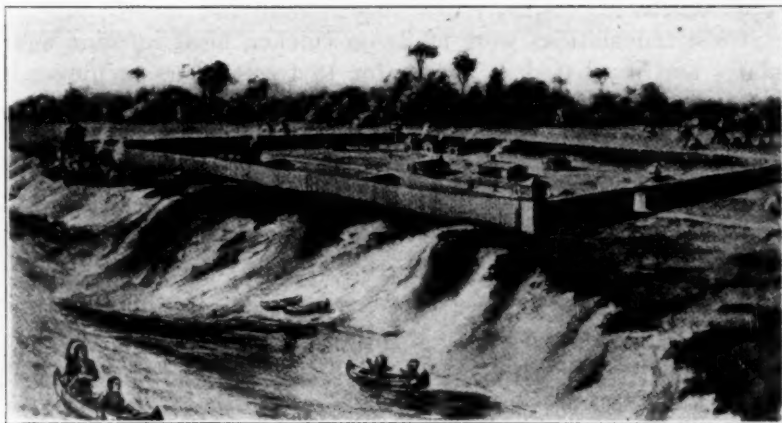
DR. KOCHENDORFER: We have these cases start in about February and they continue until say the first of April, or usually about that time, and very seldom in the fall.

You can get to the A. V. M. A. meeting in Detroit—you may not be able to go next year.

Eastern Iowa Picnic

The fourth annual veterinarians' picnic under the auspices of the Eastern Iowa Veterinary Association will be held at Riverside Park, Cedar Valley Country Club, Vinton, Iowa, Tuesday, July 9. Among the features of the occasion will be two ball games, one between the wives of veterinarians, with the blondes on one side and the brunettes on the other. This game will be umpired by Dr. J. A. Barger, of Des Moines, who will call the balls and strikes, and State Veterinarian Malcolm, who will watch the bases. Dr. E. A. Buxton, of Vinton, will act as scorekeeper. The other game will be between the Veterinarians of the North (the Cubs) and the Veterinarians of the South (the Sox) captained by Dr. Fred Williams, of Duncombe, and Dr. E. A. Horner, of Brighton, respectively. Horseshoe pitching, dancing, a band concert and golf will be other features. All veterinarians are invited to be on hand early and—bring the family.

Michigan is washed by all the Great Lakes but one.



Fort Pontchartrain, Detroit, 225 years ago

AUTOPSY FINDINGS IN FIELD CASES OF FOWL PARALYSIS*

By K. W. NIEMANN, *Manhattan, Kansas*

INTRODUCTION

Fowl paralysis, as referred to in this article, is the complete or partial loss of motor function of the muscles of the posterior limb, or of the cervical musculature, without other outward manifestations of disease. It may or may not be accompanied by local or generalized emaciation.

During the spring and summer of 1926, there came to our attention a number of cases of fowl paralysis which were often associated with intestinal parasites. It has been suggested by various workers in poultry diseases, by extension workers, and by practical poultrymen that the condition seemed somewhat substantiated and we decided to pay particular attention to the lesions in these cases in order to obtain more definite information concerning this condition.

SOURCE OF MATERIAL

The cases which are summarized in this report were obtained largely from various farm flocks of Kansas and other states. No experiment birds are included.

METHOD OF EXAMINATION

Bacteriological examination was made of all specimens received. Cultures were taken from the livers of all specimens and in some cases additional cultures from the heart, ovaries and other organs.

These inoculations were made on chicken meat infusion agar plates and incubated at 37° C. for 18 to 24 hours or longer if necessary. For final diagnosis the isolated organism was inoculated into four tubes of chicken meat infusion broth made sugar-free by the action of *Cl. welchii* and to which dextrose, lactose, maltose, and sucrose to the amount of 1 per cent had been added. No further differentiation has been made.

Microscopic examination of the intestinal contents, unfortunately, has not been made in all cases. This examination was made either by direct smears without staining, or by concentration of duodenal and cecal contents by use of a 75 per cent

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sugar solution and centrifugalization. Nearly all of the examinations were made using the concentration method. Microscopic tissue study of each case was not possible.

SUMMARY OF OBSERVATIONS

Table I shows the more or less outstanding conditions which were presented in the cases studied.

These observations were made on 93 cases. It will be readily seen that several of the various conditions were observed in the same case.

Although fowl paralysis is sometimes associated with tuberculosis, it does not seem very probable that tuberculosis is a

TABLE I—Pathological conditions found and their frequency

CONDITION	CASES	%
Cecum worms	55	59.14
Tapeworms	42	45.16
Enteritis	32	34.40
Round worms	27	29.03
Heart lesions (infiltrations and inflammations) ...	12	12.90
Coccidiosis	11	11.82
Gizzard worms	11	11.82
Tumors	9	9.67
Ruptured ovum	7	7.52
Peritonitis	4	4.30
Abscesses	3	3.22
Pneumonia	3	3.22
Obesity	2	2.15
Avitaminosis A	1	1.07
Diphtheria	1	1.07
Tuberculosis	1	1.07
<i>S. pullorum</i> type A	4	4.30
<i>S. pullorum</i> type B	1	1.07
<i>P. avicida</i>	1	1.07

specific cause of paralysis, due to the comparatively low incidence in outbreaks of tuberculosis.

We have heretofore considered tumors merely as a mechanical factor in the etiology of fowl paralysis. This we might have found to be untrue had we made careful microscopic examinations in these cases.

It seems probable that obesity, avitaminosis A and avian diphtheria do not play a very important part in causing fowl paralysis since the incidence of paralysis is practically negligible in outbreaks of these diseases. We have observed that chicks suffering from experimentally produced avitaminosis A exhibit slight nervous symptoms in the latter stages of the disease. This

was evidenced by some trembling of the limbs while the affected chick was in lateral recumbency. However, we have not noted a typical paralysis.

It is somewhat difficult to determine the exact significance of abscesses and pneumonia in these cases, especially in the presence of other factors. It is possible that the significance is not very great.

It is rather doubtful if any significance should be attached to the presence of *S. pullorum* or *P. avicida* in the paralyzed birds. We have isolated *S. pullorum* from a large number of birds in which there was no paralysis. In some of the cases reported here it was present as an ovarian infection. The presence of *P. avicida* in the one specimen was probably incidental, as it can be isolated from apparently normal birds, although Stafseth and Johnson¹ found a strain which did reproduce paralysis.

Pericarditis, epicarditis and infiltrations of the myocardium have been associated by some workers with paralysis. These conditions we have grouped under the general term of "Heart lesions." One of the birds exhibited a dilatation of the heart and petechiae were observed on the heart of several but these conditions are not included under the term "Heart lesions."

REVIEW OF LITERATURE

Stafseth and Johnson¹ express the opinion that leg weakness may be a symptom of tuberculosis, sarcomatosis, bacillary white diarrhea, fowl cholera, worm infestations, nutritional disturbances and botulism. They observed most of their cases in birds between four and fourteen months of age. It is stated that the condition is sometimes associated with eye affections and blindness. One strain of *P. avicida* was isolated by them which produced leg weakness when inoculated into apparently normal birds. Coccidia were fed to twelve birds, supplied with an adequate diet, and six of these developed paralysis. None of the birds in three adjacent pens were affected. Evidence from field and experimental cases seems, in their opinion, to show coccidiosis to be the cause of range paralysis. They have noted that the coccidia seem to localize rather deeply in the mucosa of the duodenum, also that inflammation of the duodenum was always present; the sciatic nerve may be thickened, and necrotic spots and areas appeared on the liver. They felt unable to explain the latter.

The New Hampshire Experiment Station² attacked the problem from four different angles, assuming coccidiosis to be the cause of the condition. In the first phase 200 pullets were grown on ground where there had previously been coccidiosis and paralysis while 800 pullets were grown on clean ground. Among those grown on clean ground, although they developed coccidiosis, there were fewer total cases of paralysis than among those grown on infected ground. Artificially infected birds failed to develop paralysis in two months, but the infection was extremely detrimental to the health of the birds. Losses were found to be greater and production lower in houses which were cleaned monthly as compared with houses cleaned weekly, both containing birds showing evidence of infection at the time of housing. Paralytic birds placed in coops with false bottoms which allowed the droppings to pass through failed to recover from the disease except in two cases. By way of conclusion it is stated:

The data seem to show that coccidiosis is an extremely detrimental factor in the producing of good healthy stock and that measures outlined to combat or control coccidiosis assist in controlling paralysis also.

May et al³ studied the disease in 38 Rhode Island flocks and a number of autopsies were performed in the laboratory. They state that the condition appears during August, September and October, while the birds are still on the range, but is checked when the pullets are housed. Improvement and sometimes recovery was observed upon confinement and isolation of the affected individuals. Most cases were observed "on ground that had been used for poultry for a large number of years or at least where the houses were old permanent structures that had been occupied by poultry for many years." Cases were observed in both ranging and confined stock. The range soil in all cases appeared to them to be a favorable medium for the production of coccidiosis. Some of the macroscopic postmortem lesions were: kidney tubules contained urates, spleen small, intestine was frequently inflamed or showed a thickened wall. Microscopically, infiltrations of the liver and kidney were observed. Bacteriological examination apparently revealed nothing of marked significance.

The worm infestations were observed to be rather variable as compared with apparently normal birds found to be infested. Coccidia were not uniformly present in every case. Attempts to transmit the disease were unsuccessful. A case is cited where chicks hatched from eggs of an affected flock were raised on clean

ground under proper sanitary conditions and the incidence of paralysis was reduced. Following this the cooperation of 25 flock-owners was secured and chicks on these farms were raised on clean ground with some sanitary precautions. The tobacco treatment was used to keep down worm infestations. There were no cases of paralysis in any of these flocks up to the time they were placed in the laying-house.

In all but one of 13 or 14 cases of incoordination or leg weakness, H. Kotlan⁴ found *Davainea proglottina*. These birds were between six and twelve months of age. Bacteriological examination, cage exposures, and injections of tissue extracts failed to show the presence of an infectious disease. Indefinite results were obtained from attempting to demonstrate the presence of a hematoxin in *D. proglottina*.

T. van Heelsbergen⁵ believes tapeworms to be the principal cause of paralysis.

Doyle⁶ was unable to produce the condition either by feeding or injecting material from cases of paralysis. He found gross lesions in the vagus, sciatic and splanchnic nerves and microscopically observed mononuclear cellular infiltrations.

Van der Walle and Winkler-Junius,⁷ due to their success in transmission, seemed to think paralysis due to a filtrable virus.

Pappenheimer, Dunn and Cone,⁸ in suggesting that Silver Spangled Hamburgs as a breed are more susceptible to paralysis, state that there was a heavy loss in this set of birds from coccidiosis from the seventh to the ninth week. A mortality of 14 out of 27 occurred between the twelfth and twenty-eighth week. Questionnaires sent out by these investigators elicited an opinion relative to the cause of the condition from 24 out of 55 replies. Of these, 16 associated worms or coccidiosis, or both, with the condition. They state:

None of the lesions occasionally met with such as edema and atelectasis of the lung, suppurative bronchiectasis, ulceration of the intestine associated with severe coccidiosis, etc., seemed to be related to the disease under consideration.

Heart lesions (apparently similar to those we have observed) are mentioned and they have suggested that these lesions may be either atypical or due to some agent other than that causing the paralysis. Visceral tumors were found in six out of sixty paralyzed birds and it is stated that the ovary is the apparent site of the main tumor mass in all instances and that " . . . these visceral lymphomata—in spite of their apparently neoplastic

appearance are but a manifestation of the same unknown agent which brings about infiltrations of the nervous tissues." From data presented it is concluded that " . . . there is no correlation whatever between infection with *Eimeria avium* and the incidence of fowl paralysis." According to these writers the focal character of the lesions and the absence of degenerative changes in the viscera all speak an intoxication.

Beach and Davis⁹ state that paralysis is often a symptom of chronic coccidiosis.

Ross¹⁰ in his studies of tick paralysis observed mononuclear cellular infiltrations in the posterior and anterior horns. He

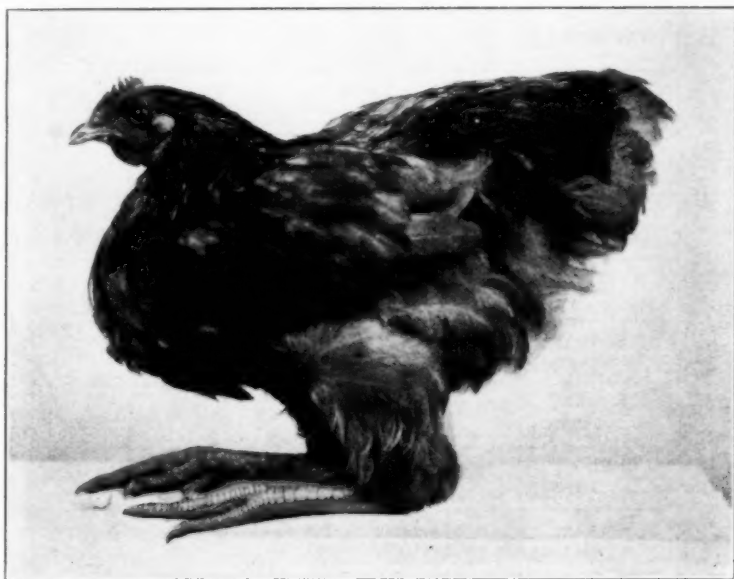


FIG. 1. Bird 26, showing paralysis due to coccidiosis

found that the peripheral nerves were not affected. A definite toxin and anticoagulin were demonstrated.

Nicolau and Galloway¹¹ found histological modifications in enzootic encephalomyelitis to be both infiltrative and degenerative.

Experiments by Ackert and Titus¹² showed that the blood-sugar content of birds infested with *A. perspicillum* was less than the control or non-infested birds.

CASE REPORTS

The autopsy findings in the cases studied are summarized in table II.

TABLE II—Summary of autopsy findings

CASE NO.	CECTM WORMS	TAP EWORMS	ENTERITIS	ROUND WORMS	HEART LESIONS	COCCID- IOSIS	GIZZARD WORMS	TUMORS	RUP TURED OVUM	PERITO- NITIS	ABSCESSES	PNEUMONIA	OBESITY	AVITAMI- NOSIS "A"	DIPH THERIA	TUBERCU- LOSIS	S. PULLO RUM TYPE A	S. PULLO RUM TYPE B	P. AVICUDA
1		++																	
2			+++++																
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4			+++++																
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19			+++++																
20			+++++																
21			+++++																

TABLE II—Summary of autopsy findings—Continued

[illegible]

DISCUSSION

The conditions which Stafseth and Johnson¹ have associated with leg weakness coincide quite well with our own observations. The cases which we have been inclined to call "range paralysis" usually appear when the birds have reached the age of twelve weeks or over. In these cases we, too, have noticed that the history has been that an outbreak typical of coccidiosis has occurred earlier in the season. The paralysis usually makes itself apparent at twelve weeks of age and then recurs at intervals or uniformly through the following winter.

Stafseth and Johnson observed eye lesions in cases of chronic coccidiosis. We have frequently observed a white, cheesy exudate in the eyes of small chicks (5-8 weeks of age) affected with coccidiosis, but have never been able to demonstrate the oöcyst in this exudate.

The liver lesions they observed are apparently the same as, or similar to, the necrotic foci which we have observed in young chicks and occasionally in older birds suffering from coccidiosis. We have been unable to demonstrate the oöcyst from these lesions.

On the whole our observations of fowl paralysis seem to be substantiated by the work and observations of these writers.

The great reduction in the incidence of the disease under sanitary conditions experienced by the New Hampshire Experiment Station would tend to show that intestinal parasitism is a factor. Whether it is a primary or secondary consideration is rather difficult to determine.

May et al³ found paralysis in range stock to be checked when the birds were housed, but from what we are able to learn, in this state the disease is not greatly reduced after the birds are housed. Perhaps the method of handling has something to do with this.

The work of Van der Walle and Winkler-Junius⁷ has not been very well substantiated by other workers.

The type of disease studied by Pappenheimer et al in their flocks of Silver Spangled Hamburgs is quite typical of the cases which we have called "range paralysis." The lesions of the respiratory tract and the intestinal ulcerations, which these writers regard as irrelevant, we are inclined to consider as possible foci for the production of toxic or other by-products which may have a peculiar affinity for the nervous system. Evidently they do not consider intestinal ulcers due to coccidiosis one of the "degenerative changes in the viscera," a lack of which they claim

all speak against an intoxication. Perhaps they had more in mind intoxication by inorganic poisons but apparently even though there is an entire absence of visceral lesions this does not exclude the absorption of a small amount of neurotoxin from the intestinal tract. The size of the wound infection in cases of tetanus are seemingly out of all proportion to the effect of the toxin and it is not unreasonable to assume that other toxins may be equally potent even though their exact nature is unknown, and that toxins may affect the nervous system alone.

Experiments by Ackert and Titus,¹² showing the blood-sugar content of parasitized birds to be less, might lead one to suspect that other changes in the composition of the blood may also take place. Often in cases of anemia in parasitized sheep the extent of the anemia is much greater than the number of parasites present would lead one to suspect.

SUMMARY

Since it has been shown by Ross¹⁰ that cellular infiltrations occur due to the presence of a specific exotoxin it would not be unreasonable to assume that the cellular infiltrations of the nervous system and viscera observed by Pappenheimer,⁸ Doyle,⁶ May,³ and Stafseth and Johnson¹ are due to neurotoxins associated with the presence of intestinal parasites particularly coccidia and tapeworms. If we select the following conditions, assuming that they are indicative of, or associated with, toxin production, we find one or more in 96.76 per cent of the 93 cases: cecum worms tapeworms, enteritis, round worms, heart lesions, coccidiosis, tumors, ruptured ovum and peritonitis.

It is undoubtedly true that all of these conditions may occur without any manifestations of paralysis. This would tend to indicate that the toxin production or absorption takes place only under certain definite circumstances.

It would appear from a review of the literature and our own observations that a toxin production occurs more readily in the presence of coccidia, tapeworms and other intestinal parasites.

A marked catarrhal enteritis and peritonitis are associated with a majority of the cases of ruptured ovum, which provides two possible locations for toxin production or absorption. However, the peritonitis is probably more of a mechanical than a septic nature and the marked catarrhal exudate usually present in the lumen of the intestine would provide a favorable place for the production of various toxins.

The possibility of a filtrable virus being the etiological factor is not to be altogether ignored, especially in the absence of definite information relative to other factors.

The great difficulty which has been experienced in reproducing the condition with any degree of regularity rather precludes the idea of any living infectious agent but does not necessarily render it an absolute improbability.

CONCLUSIONS

From our study of fowl paralysis we might draw the following conclusions:

1. The incidence of fowl paralysis in flocks previously suffering losses from coccidiosis may go as high as 50 per cent.
2. The association of tapeworms and other intestinal parasites with fowl paralysis is quite marked.
3. It seems possible that no definite single etiological factor is responsible.
4. Measures designed to control coccidiosis and intestinal parasites will greatly reduce the incidence of the disease.
5. The suggestion that the condition might be caused by more or less specific toxins associated with intestinal disturbances seems plausible.

ACKNOWLEDGMENT

We wish to express our appreciation to Dr. L. D. Bushnell, of the Department of Bacteriology, and Dr. H. F. Lienhardt, of the Department of Pathology, for many valuable suggestions used in the preparation of this paper.

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The Detroit River in August is more beautiful than the beautiful blue Danube.

STUDIES WITH BOVINE SKIN LESIONS*

By CECIL ELDER, A. M. LEE and T. R. PHELPS

University of Wyoming, Laramie, Wyoming

The skin lesion in bovine tuberculosis has of late received¹ considerable attention among workers who are interested in this disease and its control. To many it is becoming one of the most perplexing of problems in tuberculosis eradication and is furnishing material for work to many of our research men. In literature this bovine skin lesion has been referred to and known also as bovine skin tuberculosis, subcutaneous tuberculosis, skin-lesion tuberculosis, etc. In this paper we shall not spend a great deal of time upon this phase of the subject, the nomenclature, but will try only to bring the history and literature up to date for you before presenting some of the results we have obtained in our work at the University of Wyoming.

We do not believe that it is necessary in this paper to go into a detailed description of the skin lesion itself, as you all are very familiar with it, both in its appearance and its structure. One of the things which does concern us a great deal is what causes the so-called skin lesion.

Within the last few years considerable work has been done with the bovine skin lesion and at the present time there are conflicting opinions and opposite viewpoints as to the causative factors and the exact nature of this type of lesion. Some workers believe this lesion is due to an acid-fast organism other than the tubercle bacillus but capable of sensitizing an animal so that it will react to the intradermal tuberculin test. Other authorities are inclined to believe that the skin lesion is caused by the tubercle bacillus, although it may be lowered in virulence as the result of its location and residence in the skin of the bovine. Some have stated that it is a strain of the tubercle bacillus which shows a predilection for the skin and subcutaneous tissue.

Traum^{1,2} seems to be the first one to have reported this condition in cattle although he designates it under a different name, that of lymphangitis in cattle, caused by an acid-alcohol-fast organism and does not call it skin tuberculosis. In fact he did not believe at that time, according to his reports, that the condition was tuberculosis. There is little doubt in our minds

*Presented at the second annual meeting of the Intermountain Livestock Sanitary Association, Ogden, Utah, January 8-9, 1929.

but that the cases he formerly described as lymphangitis are identical with our so-called skin lesions of the present time. Other authorities agree with us in this belief as will be noted when studying the available literature upon the subject.

In 1921, Wright³ published an article entitled, "Tuberculosis Cutis," in which he made the statement: "Tuberculosis of the skin and subcutaneous tissue is apparently far more prevalent than is generally believed." He was of the opinion that tuberculosis cutis in cattle, when due to the bovine bacillus, appears for the most part in the form of tuberculosis verrucosa. To men working in this intermountainous country, particularly if they are engaged in any phase of tuberculosis control, it is hardly necessary to mention the apparently large numbers of skin lesions which are being found in this territory.

Day,⁴ in a postmortem report on tuberculin-reacting cattle, in 1921, tells about a large number of skin-lesion cases found in the western part of Wyoming in a section known as the Star Valley. Day seems to be the first one to have reported and considered skin lesions as being truly tuberculous.

Carpenter and Goldberg⁵ have made the statement that the number of skin lesions increased very materially in 1924 and 1925. They have done considerable work with the skin lesion and in their publication said: "On gross examination, these lesions simulate tuberculosis but in every case we failed to infect guinea pigs when extracts from these lesions were injected into experimental animals." These writers also believe that the lesions in the disease, described by Traum as "lymphangitis in cattle caused by an acid-alcohol-fast organism," are macroscopically and microscopically similar in all respects to those they observed in skin tuberculosis. In New York, skin lesions appear to be more prevalent in some parts of the State than others and very often they are found on the retest of herds which have passed one or more clean tests. This is the same experience as workers in the western states have reported. We have also observed that the intradermal test will pick out more reactors than any of the other tuberculin tests.

Hastings, Beach and Weber⁶ do not think skin lesions should be classed as tuberculosis. They base this conclusion, at least in part, on the fact that in skin lesions studied by them they had negative results in attempting cultures of any organism on Petroff's medium. They tried cultures both from the lesions and from material treated with antiformin. They inoculated guinea

pigs which were killed 60 to 90 days later and these showed no lesions. They found acid-fast organisms in only 7 out of 23 specimens. They concluded this disproved the possibility of the lesions being truly tuberculous.

Marsh⁷ reports his work on sixty teat-lesion cases in which he was able to find acid-fast bacilli in 45 out of 60 cases (75 per cent). Animals inoculated experimentally and cultures attempted were in general negative. He is of the opinion that these teat lesions are the same as skin lesions referred to by various other workers. He believes teat lesions are true tuberculosis, modified by its localization in the subcutaneous tissue. We are of the opinion that skin lesions and teat lesions are the same type of lesion, except for the difference in location.

Day,⁸ in a publication of March, 1928, states that the percentage of skin lesions varies greatly and he is of the opinion from his observations that this is in direct proportion to the sanitary conditions under which the cattle are kept. He thinks manure caking on the skin of the legs produces irritation and inflammation which leads to the formation of fissures in the skin through which the tubercle bacilli gain entrance from infected manure. Undoubtedly it is true and we all accept the fact that all who have studied and examined skin lesions microscopically have been able to find acid-fast bacilli which resemble those found in tuberculosis.

According to Day, at least 60 per cent of the lesions examined microscopically in his laboratory have shown acid-fast bacilli.

Day has done considerable work on the skin lesion and he has found that guinea pigs injected with suspensions from skin lesions and killed after three-month intervals were usually negative for lesions of tuberculosis. Guinea pigs treated in the same way and held five months showed a different condition. Some of these animals had enlarged lymph-nodes and at autopsy typical lesions of tuberculosis were found, not only in the enlarged lymph-nodes, but in the liver and spleen as well, in which acid-fast bacilli were found. In some cases enlarged lymph-nodes were not detected in the injected guinea pigs until six or seven months after inoculation and these also revealed tuberculous lesions. He did not state in how many guinea pigs he had been able to produce lesions of tuberculosis.

Ten calves and four pigs were injected by him with suspensions from skin lesions. On subsequent tuberculin tests none of these reacted and upon slaughter none showed lesions except one calf.

This calf reacted twice and had a tubercular lesion in the anterior mediastinal lymph-node. This lesion contained a large number of acid-fast bacilli. From this finding Day is of the opinion that the skin lesion in the cow from which the original material was obtained, was caused by bacilli of the bovine type.

Day believes that all skin lesions found in cattle that give a positive reaction to the tuberculin test, and in which acid-fast bacilli are found, are tuberculous lesions caused by the *Mycobacterium tuberculosis* which has gained entrance in the skin through abrasions, because we know of no other acid-fast organism capable of producing such skin lesions, and at the same time giving a positive reaction to tuberculin. He recognizes the fact, however, that many doubt that skin lesions are caused by *Mycobacterium tuberculosis*.

Crawford⁹ reported in June, 1928, on work with six cattle in which he tried to produce skin lesions. He used six cultures: *B. phlei*, mist bacillus, "hog skin" bacillus, avian, human and bovine tubercle bacilli. His results prove little except in that case the material used by him was incapable of producing skin lesions. The bovine bacilli produced internal lesions but no skin lesions although material was injected into the skin and beneath the skin. He worked with 20 skin lesions and failed to produce lesions upon sub-inoculation into guinea pigs, rabbits, mice, rats, chickens, or cattle. He did not state the time that the inoculated animals were carried on experiment. From this he concluded (1) that the bacteria in these skin lesions were either dead or attenuated, or (2) that a special degree of susceptibility, either inherent or artificially produced, must be present in affected cattle. Crawford states in his article that microscopic examination of over 20 subcutaneous lesions showed presence of acid-fast organisms in the majority of cases. All attempts at cultures and animal inoculations failed. Mitchell,¹⁰ in an article published in August, 1928, states that he thinks the view that skin lesions are not caused by the tubercle bacillus is unsound when it is based on the reason that one has not succeeded in infecting laboratory animals or in making isolations. He laid down the following hypotheses, assuming the acid-fast bacilli present had caused the subcutaneous lesions.

- (A) A strain of tubercle bacilli that had become altered in pathogenicity through its residence in the skin.
- (B) A strain of tubercle bacilli not conforming to any known type.

- (C) An organism separate and distinct from tubercle bacilli but capable of exciting sensitiveness to tuberculin.

According to his report, they tried to infect laboratory animals experimentally but failed in every case except in one rabbit which died in 131 days. Recently, I believe he has been able, also, to infect a guinea pig in 130 days after inoculation. They carried the material from the rabbit through seven sub-inoculations and from one of the sub-inoculations were able to culture an acid-fast organism on egg media. Mitchell formerly thought that skin lesions were not tuberculosis but a closely related organism and the reaction was in the nature of a group reaction and consequently less marked. He does not accept this interpretation now, and looks upon the lesions as truly tuberculous, although possibly caused by a strain somewhat atypical in its selection of unusual tissue for colonization.

He infected laboratory animals from only 2 out of 33 natural clinical cases of subcutaneous tuberculosis and it is interesting to note that both of these were from young animals. Mitchell was of the opinion that the age factor was important in that the animals were young and the organisms had not been present long enough to become attenuated. In older lesions he believes the organisms die.

A year ago one of the authors (Elder)¹¹ reported that up to that time our results from animal inoculation had been negative insofar as we had carried our work. Stiles¹² reported work on 79 cases studied in the Denver laboratory in which experiment animals, mostly guinea pigs, we believe, were inoculated. He was able to produce generalized tuberculosis in one guinea pig. Two guinea pigs had local abscesses, in which he demonstrated acid-fast organisms. Daines,¹³ reporting in 1928, stated that he had been able, in his work, to get one guinea-pig lesion.

For the most part, and we believe you will agree with us, infection of animals experimentally, with the subsequent production of lesions, has been more or less generally negative, at least many of us held that opinion only a year ago. Many also rather questioned the skin-lesion case as being tuberculosis. You will note from the work we are reporting today, as published during the past year, that recent findings lean towards the support of the viewpoint that skin lesions are caused by tubercle bacilli.

EXPERIMENTAL WORK

At the Wyoming Station we have worked with 35 lesions, comprising 32 skin lesions and two teat lesions and including one

TABLE I—*Tabulation of experimental work*

Lesion	Nature and Location	Animals Injected	Date	Quantity	Tuberculin	Date Died	Days	Findings	Microscopic Examination for Acid-Fast Bacilli
II	Skin (Left hock)	G.P. 22	12-23-27	P. 0.25 cc		Alive	133	Generalized intestinal tuberculosis No lesions No lesions Egg-bound	Pos. Neg. Neg. Neg.
		R. 1	12-23-27	P. 0.5 cc		5- 4-28			
		C. 14	12-23-27	E. 1 cc		K. 11-30-28			
		C. 1007	12-23-27	E. 1 cc		K. 11-30-28			
IV	Skin (Leg. Same animal as V)	C. 1157	12-23-27	Fed		9-27-28		Generalized intestinal tuberculosis	Pos.
		G.P. 45	2- 7-28	E. 0.05 cc		Alive	298		
		R. 3	2- 7-28	E. 0.1 cc		12- 1-28			
		C. 1086	2- 7-28	E. 1 cc	{ A 1 cc 2-7-28	Alive			
V	Cervical lymph-gland	C. 1296	2- 7-28	Fed	{ B 2 cc 4-21-28	Alive		Generalized tuberculosis Generalized tuberculosis Crop-bound No lesions	Pos. Pos. Neg. Neg.
		G.P. 35	2- 7-28	E. 0.1 cc		4-19-28	72		
		R. 4	2- 7-28	E. 0.1 cc		5-13-28	96		
		C. 242	2- 7-28	E. 1 cc		11- 8-28			
VII	Skin left shoulder)	C. 1366	2- 7-28	Fed		K. 11-30-28		Generalized tuberculosis No lesions No lesions	Pos. Neg. Neg.
		G.P. 28	2- 7-28	E. 0.1 cc		Alive	288		
		R. 6	2- 7-28	E. 0.1 cc		11-21-28			
		C. 166	2- 7-28	E. 1 cc		K. 11-30-28			
VII	Skin left shoulder)	C. RR	2- 7-28	Fed		K. 11-30-28		Generalized tuberculosis No lesions No lesions	Pos. Neg. Neg.
		G.P. 28	2- 7-28	E. 0.1 cc		Alive	288		
		R. 6	2- 7-28	E. 0.1 cc		11-21-28			
		C. 166	2- 7-28	E. 1 cc		K. 11-30-28			

Pos.
Neg.
Neg.

No lesions
No lesions

K. 11-30-28

XI	Skin (left fore leg)	G.P. M G.P. 50 R. 13 R. 14 C. 2138 C. 2223 C. 2372 C. 2362 C. 2100 C. 2272	2- 9-28 2- 9-28 2- 9-28 2- 9-28 2- 9-28 2- 9-28 2- 9-28 2- 9-28 2- 9-28 2- 9-28	P. 0.25 cc P. 0.25 cc P. 0.5 cc P. 0.5 cc E. 1 cc E. 1 cc E. 1 cc E. 1 cc Fed Fed	B 2 cc 3- 6-28 A 1 cc 3-26-28 B 3 cc 3-26-28 A 1 cc 3-26-28	3-26-28 9-24-28 5-14-28 9-26-28 K. 11-30-28 K. 11-30-28 11-25-28 11-30-28 K. 11-30-28 K. 11-30-28	230	Animal missing No lesions Fibrous pleuro-pneumonia Generalized tuberculosis No lesions No lesions Abdominal tumors No lesions No lesions No lesions	Neg. Neg. Pos. Neg. Neg. Neg. Neg. Neg. Neg.
XX	Skin (hock)	G.P. 61 R. 66 C. 286 C. 200	4-21-28 4-21-28 4-21-28 4-25-28	E. 0.05 cc E. 0.1 cc E. 2 cc Fed	B 1 cc 4-21-28 B 2 cc 5-31-28 B 1 cc 4-21-28 B 2 cc 5-31-28 B 2 cc 4-21-28 B 3 cc 5-31-28 B 2 cc 4-25-28 B 3 cc 5-31-28	6- 1-28 9-28-28 Alive Alive	41 160	Tuberculosis of mesentery Generalized tuberculosis	Pos. Pos.
XXV	Skin (stifle and cervical)	G.P. 83 R. 73 C. 350y C. 2414	5-15-28 4-21-28 4-21-28 4-21-28	P. 0.25 cc P. 0.5 cc P. 2 cc Fed	B 1 cc 5-15-28 B 2 cc 6-20-28 B 1 cc 4-21-28 B 2 cc 5-31-28 B 2 cc 4-21-28 B 3 cc 5-31-28 B 2 cc 4-21-28 B 3 cc 5-31-28	Alive Alive 12- 4-28 Alive	227	Generalized tuberculosis	Pos.
XXX	Teat	G.P. 86 R. 70 C. 2396	6- 6-28 6- 6-28 6- 6-28	E. 0.05 cc E. 0.1 cc E. 2 cc	B 1 cc 6- 6-28 B 2 cc 7-13-28 B 1 cc 6- 6-28 A 1 cc 6- 6-28	11-12-28 Alive Alive	159	Generalized tuberculosis	Pos.

TABLE II—*Tabulation of experimental work*

LESION	NATURE AND LOCATION	ANIMALS INJECTED	DATE	QUANTITY	TUBERCULIN	DATE DIED	FINDINGS	MICROSCOPIC EXAMINATION FOR ACID-FAST BACILLI
I	Skin	G.P. 43	11- 4-27	P. 0.25 cc		Alive	Negative	Neg.
		R.D.	10-28-27	E. 0.5 cc		Missing		
		C. 1149	10-28-27	E. 1 cc		K. 11-30-28	Double ocular roup	Neg.
		C. 1387	10-28-27	E. 1 cc		K. 11-30-28	Double ocular roup	Neg.
III		C. 1301	10-28-27	Fed				
		G.P. 44	2- 4-28	E. 0.125 cc		Alive		
		R. 2	2- 4-28	E. 0.125 cc		Alive		
		C. 1023	2- 4-28	E. 1 cc		Alive		
VI	Skin (left shank)	C. 230	2- 4-28	E. 1 cc		Alive	Enteritis and paralysis	Neg.
		C. 8	2- 4-28	Fed		5- 7-28		
		G.P. 27	2- 7-28	E. 0.05 cc		10-24-28	No lesions	Neg.
		R. 5	2- 7-28	E. 0.1 cc		4-15-28	Broken back	Neg.
VIII	Skin (left fore leg)	C. 18	2- 7-28	E. 1 cc		5-18-28	Abdominal tumors	Neg.
		C. 234	2- 7-28	Fed		Alive		
		G.P. 30	2- 9-28	E. 0.05 cc		Alive		
		G.P. 47	2- 9-28	E. 0.05 cc	B 1 cc 2-9-28	Alive	Broken back	Neg.
		R. 96	2- 9-28	E. 0.1 cc	B 1 cc 2-9-28	7-30-28	Injury	Neg.
		R. 84	2- 9-28	E. 0.1 cc	B 3 cc 2-9-28	7-15-28		
		C. 2116	2- 9-28	E. 1 cc	B 3 cc 2-9-28	Alive		
		C. 2172	2- 9-28	E. 1 cc	B 3 cc 2-9-28	Alive		
		C. 1449	2- 9-28	E. 1 cc		7-30-28	Double ocular roup	Neg.
		C. 2076	2- 9-28	E. 1 cc		12- 6-28	Pullorum infection	Neg.
		C. 1500	2- 9-28	Fed		5-15-28	Tumors and paralysis	Neg.
		C. 2092	2- 9-28	Fed	B 3 cc 2-9-28	10- 8-28	No lesions	Neg.

Neg.
Neg.
Neg.

12- 6-28
5-15-28
10- 8-28
Pullorum infection
Tumors and paralysis
No lesions

B 3 cc 2-9-28

Fed
Fed

2- 9-28
2- 9-28

C. 1500
C. 2092

IX	Skin (left shoulder)	G.P. 37	2- 9-28	E. 0.05 cc	B 1 cc 2-9-28	10-19-28	Pneumonia Congestion of lungs	Neg. Neg.
		G.P. 99	2- 9-28	E. 0.05 cc	B 1 cc 2-9-28	11-16-28		
		R. 9	2- 9-28	E. 0.1 cc	B 1 cc 2-9-28	Alive		
		R. 56	2- 9-28	E. 0.1 cc	B 3 cc 2-9-28	Alive		
X	Skin (left hock)	C. 2391	2- 9-28	E. 1 cc	B 3 cc 2-9-28	K. 11-30-28	No lesions	Neg.
		C. 2049	2- 9-28	E. 1 cc	B 3 cc 2-9-28	Alive		
		C. 2407	2- 9-28	E. 1 cc	B 3 cc 2-9-28	Alive		
		C. 2050	2- 9-28	E. 1 cc	B 3 cc 2-9-28	Alive		
		C. 2437	2- 9-28	E. 1 cc	B 3 cc 2-9-28	Alive		
		C. 2379	2- 9-28	Fed	B 3 cc 2-9-28	Alive		
		G.P. 31	2- 9-28	P. 0.25 cc	A 1 cc 3-26-28	5- 3-28		
		G.P. 34	2- 9-28	P. 0.25 cc	A 1 cc 3-26-28	9-21-28		
		R. 11	2- 9-28	P. 0.5 cc	B 3 cc 3-26-28	7-27-28		
		R. 12	2- 9-28	P. 0.5 cc	A 1 cc 3-26-28	9-28-28		
XII		C. 2422	2- 9-28	E. 1 cc	B 3 cc 3-26-28	Alive	Fibrinous peritonitis Metritis and hepatitis Necrophorus infection Pneumonia	Neg. Neg. Neg. Neg.
		C. 2326	2- 9-28	E. 1 cc	A 1 cc 3-26-28	Alive		
		C. 2434	2- 9-28	E. 1 cc	B 3 cc 3-26-28	Alive		
		C. 2427	2- 9-28	E. 1 cc	B 3 cc 3-26-28	Alive		
XIII		C. 1450	2- 9-28	Fed	B 3 cc 3-26-28	Alive	Hepatitis and metritis Injury	Neg. Neg.
		C. 2111	2- 9-28	Fed	B 3 cc 3-26-28	Alive		
		G.P. 41	2-18-28	P. 0.25 cc	B 1 cc 2-18-28	11- 1-28		
		R. 15	2-18-28	P. 0.5 cc	B 1 cc 2-18-28	12-25-28		
		C. 2279	2-18-28	E. 1 cc	B 3 cc 2-18-28	Alive	No lesions	Neg.
		C. 2101	2-18-28	Fed	B 3 cc 2-18-28	Alive		
		G.P. 36	2-18-28	P. 0.25 cc	B 1 cc 2-18-28	9-21-28		
		R. 98	2-18-28	P. 0.5 cc	B 1 cc 2-18-28	Alive		
		C. 2124	2-18-28	E. 1 cc	B 3 cc 2-18-28	Alive	Abdominal tumor	Neg.
		C. 2239	2-18-28	Fed	B 3 cc 2-18-28	6-16-28		

TABLE II—*Tabulation of experimental work—Continued*

Lesion	Nature and Location	Animals Injected	Date	Quantity	Tuberculin	Date Died	Findings	Microscopic Examination for Acid-Fast Bacilli
XIV		G.P. 46	2-18-28	P. 0.25 cc	B 1 cc 2-18-28	Alive	Bitten through back Tumors in liver	Neg.
		R. 17	2-18-28	P. 0.5 cc	B 1 cc 2-18-28	Alive		Neg.
		C. 2227	2-18-28	E. 1 cc	B 3 cc 2-18-28	Alive		Neg.
		C. 1128	2-18-28	Fed	B 3 cc 2-18-28	12-21-28		Neg.
XV		G.P. 39	2-18-28	E. 0.05 cc	B 2 cc 3-26-28	Alive	Septic metritis Roup	Neg.
		R. 94	2-18-28	E. 0.1 cc	A 1 cc 3-26-28	Alive		Neg.
		C. 1275	2-18-28	E. 1 cc	A 1 cc 3-26-28	9- 1-28		Neg.
		C. 2023	2-18-28	Fed	B 3 cc 3-26-28	Alive		Neg.
XVI		G.P. 42	2-18-28	E. 0.05 cc	A 1 cc 3-26-28	Alive	Injury fighting Fatty degeneration of liver	Neg. Neg.
		R. 19	2-18-28	E. 0.1 cc	B 2 cc 3-26-28	12-31-28		
		C. 2015	2-18-28	E. 1 cc	A 1 cc 3-26-28	7-11-28		
		C. 2226	2-18-28	Fed	B 3 cc 3-26-28	Alive		
XVII*		G.P. 38	2-18-28	E. 0.05 cc	B 2 cc 3-26-28	11- 5-28	Animal missing	
		R. 68	2-18-28	E. 0.1 cc	B 2 cc 3-26-28	Alive		
		C. 1240	2-18-28	E. 1 cc	A 1 cc 3-26-28	Alive		
		C. 2359	2-18-28	Fed	B 3 cc 3-26-28	Alive		
XVIII	Skin (used cervical present)	G.P. 51	4-21-28	E. 0.05 cc	A 1 cc 4-21-28	12- 5-28	Lobar pneumonia Paralysis	Neg. Neg.
		R. 52	4- 5-28	E. 0.1 cc	A 1 cc 4- 5-28	Alive		
		C. 1129	4- 5-28	E. 2 cc	A 1 cc 4- 7-28	Alive		
		C. 1045	4- 5-28	Fed	A 1 cc 4- 7-28	5- 4-28		
			9- 2-28				Congestion of lungs	Neg.

					A 1 cc 4-7-28	5-4-28	Paralysis	Neg.
XIX	Skin (cervical region)	G.P. 60 R. 65 C. 2415 C. 2405	4-21-28 4-21-28 4-21-28 4-21-28	E. 0.05 cc E. 0.1 cc E. 2 cc Fed		9-2-28 Alive 6-14-28 Alive	Congestion of lungs No lesions	Neg.
								Neg.
								Neg.
XXI*	Skin (abdom ² me.)	G.P. 82 R. 64 C. 278 C. 204	4-21-28 4-21-28 4-21-28 4-21-28	E. 0.05 cc E. 0.1 cc E. 2 cc Fed	B 1 cc 4-21-28 B 2 cc 5-31-28 B 1 cc 4-21-28 B 2 cc 5-31-28 B 2 cc 4-21-28 B 3 cc 5-31-28 B 2 cc 4-21-28 B 3 cc 5-31-28	11-14-28 9-29-28 Alive 10-24-28	No lesions Peritonitis and hepatitis No lesions	Neg.
								Neg.
								Neg.
XXII	Skin (hock and knee)	G.P. 58 G.P. 76 R. 93 C. 209 C. 123	4-21-28 5-15-28 4-21-28 4-21-28 4-21-28	E. 0.05 cc P. 0.25 cc E. 0.1 cc E. 2 cc Fed	A 1 cc 6-20-28 A 1 cc 5-31-28 A 1 cc 4-21-28 A 2 cc 5-31-28 A 2 cc 5-31-28	4-26-28 11-5-28 9-28-28 Alive Alive	Septicemia No lesions-pregnant Lobar pneumonia	Neg.
								Neg.
								Neg.
XXIII	Skin (knee)	G.P. 59 R. 75 C. 130 C. 274	4-21-28 4-21-28 4-21-28 4-21-28	E. 0.05 cc E. 0.1 cc E. 2 cc Fed	A 1 cc 5-31-28 A 1 cc 5-31-28 A 2 cc 5-31-28	Alive Alive 6-7-28 Alive	Missing	
XXIV	Skin (hock)	G.P. 77 R. 72 C. 77 C. 167	5-15-28 4-21-28 4-25-28 4-25-28	P. 0.25 cc P. 0.5 cc P. 2 cc Fed	A 1 cc 6-20-28 A 1 cc 5-31-28 A 2 cc 5-31-28 A 2 cc 5-31-28	9-13-28 Alive 6-3-28 7-5-28	Purulent hepatitis Double ocular roup Obesity	Neg.
								Neg.
								Neg.
XXVI	Skin (shoulder and cervical)	G.P. 79 R. 74 C. 1481 C. 114	5-15-28 4-21-28 4-21-28 4-21-28	P. 0.25 cc P. 0.5 cc P. 2 cc Fed	B 1 cc 5-15-28 B 2 cc 6-20-28 B 1 cc 4-21-28 B 2 cc 5-31-28 B 2 cc 4-21-28 B 3 cc 5-31-28 B 2 cc 4-21-28 B 3 cc 5-31-28	9-22-28 Alive 6-5-28 Alive	No lesions Double ocular roup	Neg.
								Neg.
								Neg.

TABLE II—*Tabulation of experimental work—Continued*

Lesion	Nature and Location	Animals Injected	Date	Quantity	Tuberculin	Date Died	Findings	Microscopic Examination for Acid-Fast Bacilli
XXVII	Skin (knee and shoulder)	G. P. 80	5-15-28	P. 0.25 cc	B 1 cc 5-15-28	Alive		
		R. 71	4-21-28	P. 0.5 cc	B 1 cc 4-21-28	Alive		
		C. 191	4-21-28	P. 2 cc	B 2 cc 4-21-28	Alive		
		C. 277	4-21-28	Fed	B 2 cc 4-21-28	Alive		
XXVIII	Skin (knee and pre-scapular)	G. P. 81	5-21-28	P. 0.125 cc	A 1 cc 6-25-28	Alive		
		R. 55	6- 1-28	P. 0.25 cc	A 1 cc 6- 1-28	Alive		
		R. 67	4-21-28	P. 0.5 cc	A 1 cc 7- 6-28	5- 5-28		
		C. 1495	4-21-28	P. 2 cc	A 1 cc 4-21-28	Alive	Killed fighting	Neg.
		C. 2417	4-21-28	Fed	A 2 cc 5-31-28	Alive		
XXIX	Teat	G. P. 85	6- 6-28	E. 0.05 cc	B 1 cc 6- 6-28	Alive		
		R. 69	6- 6-28	E. 0.1 cc	B 2 cc 7-13-28	Alive		
		C. 116	6- 6-28	E. 2 cc	B 1 cc 6- 6-28 B 2 cc 7-13-28 A 1 cc 6- 6-28 A 2 cc 7-13-28	Alive		
XXXI	Skin	G. P. 89	7-13-28	P. 0.25 cc	B 1 cc 7-13-28	8-14-28	Fibro-purulent perihepatitis	Neg.
		R. 88	7-13-28	P. 0.25 cc	B 1 cc 8-10-28	8-25-28	Necrophorus infection	Neg.
		C. 2289	7-13-28	P. 2 cc	B 1 cc 7-13-28	Alive		
		C. 2280	7-13-28	Fed	B 2 cc 8-10-28 A 2 cc 8-10-28			

XXXII*	Skin	G.P. 90	7-13-28	P. 0.25 cc	B 1 cc 7-13-28 B 1 cc 8-10-28 B 1 cc 7-13-28 B 2 cc 8-10-28 A 2 cc 8-10-28	Alive Alive Alive		
XXXIII		R. 87	7-13-28	P. 0.5 cc			No lesions	Neg.
		C. 2424	7-13-28	P. 2.5 cc			Ear mange	Neg.
		C. 2424	7-13-28	Fed				
		G.P. 97	8- 6-28	P. 0.125 cc	B 1 cc 8- 6-28 B 2 cc 9-22-28 B 1 cc 8- 6-28 B 2 cc 8- 6-28	11-15-28 9- 1-28 Alive		
XXXIV		R. 91	8- 6-28	P. 0.25 cc				
		C. 1152	8- 6-28	P. 1 cc				
		C. 1152	8- 6-28	Fed				
		G.P. D31	9-22-28	P. 0.25 cc	B 1 cc 9-22-28 B 1 cc 9-22-28 B 2 cc 9-22-28	Alive Alive Alive		
XXXV		R. 100	9-22-28	P. 0.25 cc				
		C.B.R.	9-22-28	P. 2 cc				
		C.B.R.	9-22-28	Fed				
	Skin (left fore leg)	Acid-fast organisms present but not enough material for injecting.						

*Negative for acid-fast bacilli.
November 28, 1928: C. 1086—C. 1296—C. 1366, negative tuberculin test.
G. P. 58 replaced by G. P. 76.
R. 67 replaced by R. 55.

skin lesion from a carcass which also showed a cervical lesion and upon which we are reporting. We thought you might be interested in a tabulation and summary of our work up to the present time.

We have received the skin lesions packed in borax and also in fresh state and have handled them in the following manner. Smears on microscopic slides were made direct from a small portion of the lesion. Parts of the lesion, or, in cases where the lesions were small, the whole lesions, were ground up with physiological salt solution in a sterile mortar. In some cases this suspension was injected directly into experiment animals. In other cases we treated the skin lesions by Petroff's method and injected the treated material into experiment animals. In our work we used guinea pigs, rabbits and chickens. The guinea pigs and rabbits were injected intraperitoneally. The chickens were divided; some injected intraperitoneally, some fed parts of ground-up lesions, and a few were both fed and injected intraperitoneally. The accompanying tables will show the dates, amounts injected, and results as well as types of lesions used.

KEY TO TABLES

For the most part the tables are self-explanatory. Under the columns headed "Quantity," E means emulsion or suspension (lesions ground up in salt solution). P means material treated by Petroff's method. In column headed "Tuberculin," A refers to avian and B to bovine tuberculin. Under column "Date died," K means that the animal was killed.

DISCUSSION OF TABLES

There are included in table I only the lesions from which we got positive results upon animal inoculation in one or more of the experiment animals used. Table II includes lesions upon which we are reporting negative findings at the present time. You will note that the guinea pig and rabbit on the lymph-gland lesion V died of generalized tuberculosis in 72 days and 96 days respectively, and the skin lesion from the same animal produced tuberculosis in rabbit 3. In all, eight positive lesions were reported but one of them is a lymph-gland from which we should expect positive findings upon animal inoculation. Seven skin lesions out of 33 skin lesions studied (21.2 per cent) have given us positive results, which is a much higher percentage than most laboratory workers are reporting. The 33 skin lesions include two teat lesions. It should be kept in mind that we are not re-

porting a complete study, as our work is not finished. There may be more animals die from lesions which we are reporting negative today, due to the fact that they have not been carried long enough.

As a general rule the period of incubation in experiment animals is long, an average of 192 days, leaving out animals upon the lymph-gland lesion. We have been able to produce tuberculous lesions in five rabbits, two guinea pigs, and one chicken. We hardly know how to account for tuberculosis in the chicken, except that the material injected produced the condition. All birds used were mature hens from our University flock in which we have never found a tuberculous bird. They were all negative to the tuberculin test before being used on this experiment and have been kept in a strict quarantine the entire time.

We believe that our work proves that one-fifth of the skin lesions with which we worked contained organisms capable of producing tuberculous lesions in experiment animals, and that even though they may have been somewhat attenuated they were alive and had some virulence.

We have examined smears direct from all the skin lesions which we received and have found acid-fast bacilli in 91.1 per cent of the lesions. We have called lesions positive for acid-fast bacilli only when we found acid-fast rods. Acid-fast granules have been observed in many of our slides. In October we reported in an unpublished report that we had found acid-fast bacilli in 55 per cent of the skin lesions examined by us. The last month we have re-examined our slides and after long diligent searches and researches have found acid-fast bacilli in 31 out of the 34 lesions examined. We feel that if one would examine all the material in a lesion and look long enough that you could find acid-fast organisms in practically 100 per cent of the skin lesions. All of the lesions reported in the first table have revealed the presence of acid-fast bacilli in smears made direct from the lesions.

From the tables included in this report you will note we have used avian and bovine tuberculins on some of our experiment animals, sometimes at time of injection with original material, sometimes several weeks later, and in other cases at both times. Tables will show the kind, amounts and dates used. Tuberculin was used to see if it would break down the animals' resistance or aid the organisms present in the skin lesions in getting a foothold and producing disease. You will note that one animal, guinea pig

61, died soon after the injection of tuberculin. This may be only a coincidence or may have some significance but we are of the opinion that the use of tuberculin had little bearing on the production of experimental lesions.

SUMMARY

1. Work was done with 35 tubercular lesions of which 32 were skin lesions, two were teat lesions, and one was cervical lymph-gland.
2. Injections were made into guinea pigs, rabbits and chickens with all these lesions except one of the skin lesions.
3. Six of the skin lesions, one of the teat lesions, and the one cervical lesion produced tuberculosis in some of the experiment animals.
4. Twenty-one per cent of the skin lesions, including one teat lesion, produced tuberculosis in experiment animals.
5. The period of incubation (average, 192 days) was long, illustrating that the organisms in the skin lesion were of low virulence as compared with the usual bovine tubercle bacilli.
6. Acid-fast bacilli were found in 91.1 per cent of the lesions studied.
7. All the experiment animals reported tuberculous died, none being killed. Many experiment animals injected with skin lesions are still living.
8. Tuberculin tests have been run upon experiment animals but none have reacted. Both avian and bovine tuberculins have been used.

ACKNOWLEDGMENTS

We wish to take this opportunity to thank Dr. W. E. Howe, Bureau of Animal Industry inspector-in-charge at Denver, who has furnished us with most of the skin lesions which we have used in this work, and for his helpful suggestions.

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The Southeastern Michigan Veterinary Medical Association, with headquarters at Detroit, claims to be the livest local veterinary association on the map.

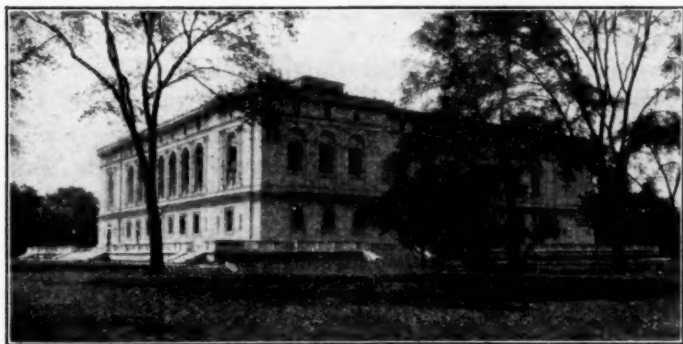
Maryland Meeting

The announcement of the summer meeting of the Maryland State Veterinary Medical Association as given in the June issue of the JOURNAL was incorrect. The meeting will be held at College Park, instead of Baltimore, and the correct dates are July 18 and 19, instead of 19 and 20.

Violators Successfully Prosecuted

Dr. William Hilton, secretary-treasurer and registrar of the Veterinary Association of Manitoba, reports two successful prosecutions of violations of the Manitoba Veterinary Practice Act recently. L. McArthur, of Selkirk, was fined \$20 and costs and George Horell, of Benito, was fined the same amount and costs, for illegally practicing as veterinary surgeons in their respective communities. In the first case the prosecution was conducted by the provincial police of Winnipeg and in the other by the provincial police of Swan River.

Michigan's good roads lead to Detroit.



Public Library, Detroit

MILK INSPECTION IN A SMALL TOWN*

By W. H. HILTS, *Milk Inspector,*

Elko, Nevada

For the past six years, the City of Elko, Nevada, a small town of about 3,000 population, has maintained a regular system of milk inspection. Previous to 1922, there was no regular inspection. Once or twice a year, an inspection was made by the State Department of Pure Food and Drugs. This inspection helped to some extent but was not frequent enough to be adequate. The quality of the milk at that time is not known to the writer but he has been told that sour milk was not uncommon, complaints were many, customer turn-over was large and consumption of milk was small, especially in the summer months that are less favorable for good milk. Since regular milk and dairy inspection has been established, all this is a thing of the past.

The event directly responsible for the establishment of milk inspection in Elko was the finding of sixteen tuberculous cows in a herd of twenty-three cows supplying the city with raw milk. This event forcibly impressed upon the people of the city the necessity for frequent and constant supervision of the milk supply. It so happens that one of the milk distributors of the city is a graduate veterinarian. By reason of his knowledge of both the veterinary profession and the dairy business, he is unusually well posted on the many advantages of milk inspection. As a veterinarian he knew that milk inspection would protect the health of the people to a great extent and as a milk distributor he knew that anything that built up the quality of the milk would also build up his own business. He recognized that the finding of this large number of tuberculous cows had focused the attention of the people on their milk supply and that conditions were ripe to start agitation for city milk inspection. Largely through his own individual efforts, a model ordinance was procured from the State Department of Pure Food and Drugs, submitted to the City Council and promptly passed. Immediately following passage of the ordinance, arrangements were made with the State Board of Stock Commissioners to have the writer, who is an employe of that Board and stationed at Elko, take on the work. It was estimated that the work would require

*Presented at the second annual meeting of the Intermountain Live Stock Sanitary Association, Ogden, Utah, January 8-9, 1929.

about two days per month and the City agreed to pay the Board \$25.00 per month for the service. Equipment was purchased, arrangements were made to do the milk-testing in the laboratory of the County hospital, where a microscope and incubator were available, and the work started. With very few exceptions it continues today as it was when it was first started.

The milk supply and the dairies are inspected at irregular intervals, usually about once a month but oftener if necessary. More attention is given to the work in the spring and fall, as these are the critical times in milk-production. Dairies are inspected and scored more or less in accord with the U. S. Department of Agriculture score-card and with the Department Circular 276, entitled, "Inspection of Milk Supplies," special attention being paid to the sterilization of milk containers and to cows' udders. Milk-rooms, entirely separated from the barns, small-top milk pails and milk-coolers are insisted upon, although the coolers are not always used during the very cold winter months when temperatures go down to zero or below. Well-ventilated and well-lighted modern barns and many other things are very desirable, but they are not considered nearly so important as the other things just mentioned. It is required that dairy cattle be tuberculin-tested annually and reactors immediately removed and slaughtered.

ROUTINE OF TESTING MILK SAMPLES

Milk samples for testing are obtained from the delivery wagons. The milk is tested for butter fat, solids not fat, total solids, added water, sediment and a bacterial estimate is made. Tests are also made for adulterations, preservatives, etc., when they are suspected. All of these tests are quite simple and can be made by any veterinarian without a great deal of special preparation or equipment. The routine of testing milk samples is as follows:

The samples are taken from the delivery wagon without any previous notice to the dairy or driver. If the sample does not feel cool enough to the touch, two samples are taken and the temperature of one of them is ascertained on the spot. The other sample is placed on ice and taken to the laboratory as soon as possible. Immediately on arrival at the laboratory, and without removing the cap, the milk is mixed as thoroughly as possible by inverting the bottle several times and by skaking it. The cap is then removed and 10 cc of milk is taken with a

sterile pipette for the bacterial estimate. For making this estimate the reductase test is used.

Formerly, the plate method was used for making bacterial counts but for several reasons this method was discontinued in favor of the reductase test. The latter is very simply made and, while it has its disadvantages, just as other methods have, still it is accurate enough for practical purposes. The technic for making this test may be found in New Hampshire Agricultural Experiment Station Circular 23, June, 1924. In addition to the reductase test, samples are examined microscopically when it is considered necessary.

The sample is then warmed to about 60° F., thoroughly mixed by pouring and the lactometer reading is taken. Then the sample is again mixed and the Babcock sample is taken. The remainder of the milk in the bottle is then forced through the sediment tester. The cotton disc containing the sediment is removed, marked and mounted. The Babcock test for butter fat is then completed. This completes the actual examination of the milk unless it is considered advisable to make further tests for preservatives, etc.

The "solids not fat" content is computed from the butter fat content and the lactometer reading by means of a percentage table that is found in Klein's *"Principles and Practice of Milk Hygiene"* and other texts.

When the lactometer and butter fat readings point toward a possibility of added water in the milk, the percentage of added water is determined by using the following formula: Lactometer reading, plus butter-fat content, multiplied by 100, divided by 34.5 equals x. One hundred minus x equals percentage of added water. This formula is not exactly correct, of course, but will detect any appreciable amount of added water.

DAIRYMAN RECEIVES CERTIFICATE

Following completion of the examination, a permanent record of the results is made and a certificate stating the results of the examination is sent to the dairyman. In case violations of the ordinance are detected, a copy of the certificate is sent to the City Clerk and immediate steps are taken to locate the cause of the trouble and to remove it. In many instances the dairyman is unaware that there is anything wrong with his milk and in such cases he very much appreciates the helpful assistance of the inspector. After the dairyman has been given a reasonable

amount of time to bring his milk back to standard, it is again tested. The second test practically always shows that the milk is back to standard. To date it has not been considered advisable to prosecute any of our dairymen for violations of the milk ordinance. Violations are frequently detected but in practically all instances they are mistakes of the hand rather than of the heart and no prosecution is indicated. We believe, however, that when a dairyman proves himself to be negligent and indifferent to the quality of his milk he should be promptly and permanently put out of the business. This may be done either through the courts or by public opinion. We prefer the court of public opinion, as our experience has twice shown us that this method works swiftly and surely in a small community. Court actions frequently only start trouble, whereas the judgment of public opinion is final.

ANY COMMUNITY CAN HAVE INSPECTION

We believe that the City of Elko, Nevada, has demonstrated that any organized community, large or small, can have regular and efficient milk inspection at small cost, provided only, that the services of a veterinarian are available.

This is about all there needs to be said at this time regarding milk inspection in Elko. But there is a great deal that may well be said about the lack of milk inspection in the other towns in this region. There is nothing surprising about what has been done in Elko. The surprising thing is that it has not been done in many other places. Probably one reason why more communities do not have milk inspection is on account of the prevailing belief that this work requires highly trained specialists and that the work is too expensive for small communities. Both of these ideas are erroneous. Any practicing veterinarian is already at least 75 per cent qualified to do the work and with very little additional study and application can easily qualify himself to do very effective work. The veterinarian is the key to the situation and where one is available, even for part-time service, the community can have regular milk inspection.

So far as the cost is concerned, it amounts to practically nothing. In Elko the annual cost to each inhabitant is about the same as the cost of a single quart bottle of milk. Certainly, 12 cents per year per person is no obstacle to regular milk inspection.

The entire benefits of milk inspection are much greater than is generally known. It benefits not only the consumer but the

dairyman and the veterinarian as well. The beneficial results as a public health measure are well known to a group of this kind and need not be repeated here but the beneficial results to the dairymen and to the veterinarian are not so well known and may well be brought out at this time.

In places where milk inspection has been established the better dairymen are always enthusiastic supporters of it, not altogether from a health standpoint but from a "better business" standpoint. Many of the larger milk distributors have milk inspectors on their own payrolls because they have learned that milk inspection leads to better milk, better milk always means more milk used and consequently more business for the dairymen. According to the last report of the Nevada State Pure Food and Drug Commissioner, Elko has the highest per capita consumption of market milk in Nevada. Dairymen also profit considerably through the advice given them by inspectors in regard to animal health and dairy sanitation. They very much appreciate the regular analysis of their milk and cream. This is particularly true of the small dairyman who is hardly in a position to make this analysis for himself. The milk inspector is the dairyman's best friend.

BENEFITS THE VETERINARIAN

The benefits to the veterinarian are just as great but for some reason or other he is not receiving these benefits. Possibly he, too, believes that milk inspection requires highly specialized training and that he is not competent to do the work. Let me repeat that this is not so. Many veterinarians received college training in milk and dairy inspection and are already qualified to do the work. Others can easily qualify themselves by the study of a few small pamphlets that can usually be obtained free from the U. S. Department of Agriculture. The practicing veterinarian who is not doing milk inspection is, in a great many instances, overlooking an opportunity to be of greater service to his community and to add a substantial increase to his income.

By this time I trust that at least some of you are wondering just how to go about it to get this kind of work started. There are many things that can be done, but the first, and most important, is to familiarize yourself as much as possible with the work, so that you will be prepared when the demand comes for your services. The demand will surely come as soon as the proper foundation has been laid. The veterinarian can be very

instrumental in laying the foundation for this demand but in most instances he should not do so openly and directly lest he be accused of trying to feather his own nest. A few well-chosen remarks here and there with dairymen and with influential citizens of the community will sometimes start the ball to rolling but the veterinarian will do well to keep himself in the background. He might contrive, through a close friend, to have himself requested to present a paper on milk inspection before women's clubs or other organizations interested in community welfare. In such a case the friend should make it plain to the members of the organization that he has requested the veterinarian to present the paper. Newspapers, county health officers and others can be of considerable assistance. Various federal and state agencies are always glad to assist in work of this kind and model ordinances can be obtained from them. A survey of the milk supply of the community, made by the proper state official or other competent outsiders, usually furnishes information that is valuable for a starter. One more suggestion: In your campaign for clean milk, while you are proclaiming the dangers and disadvantages of unclean milk, do not forget to proclaim the advantages of clean milk.

In closing, let me quote Dr. Woods Hutchinson, a physician and eminent writer on public health matters, who says:

A pure and wholesome milk supply will not come of itself, nor will it continue a moment longer than inspection is maintained. Eternal vigilance is the price of liberty from milk-borne infections, and money spent in maintaining proper milk control is the soundest of investments for it will repay itself, year after year, in the health of the growing generations.

Ladies, Plan for Detroit!

In a little more than a month, the American Veterinary Medical Association will meet in Detroit. The program for the ladies includes a boat-ride to Bois Blanc (Bob-Lo), a theatre party, a shopping tour, a card party, a sight-seeing tour and other forms of entertainment possible in a city situated as is Detroit.

A meeting of unusual interest is being planned for the Women's Auxiliary. Come with your husband to Detroit, August 13, 14, 15 and 16, and help make this meeting the largest in the history of the Association.

MRS. A. S. SCHLINGMAN,

*Chairman, Sub-Committee on Reception and
Entertainment of Ladies.*

FURTHER MISCELLANEOUS TESTS OF TETRACHLORETHYLENE AS AN ANTHELMINTIC

By A. S. SCHLINGMAN, *Detroit, Michigan.*

Medical Research Laboratories, Parke, Davis and Company

Soon after Hall and Shillinger¹ recommended the use of tetrachlorethylene as an anthelmintic for dogs, experiments were begun by the author to compare the action of this drug with that of carbon tetrachlorid, a somewhat similar chlorin compound, which had been used for this purpose for some years.

It was found that tetrachlorethylene was exceptionally efficient against the roundworms and hookworms of these animals and was safer to administer, especially to puppies.²

Later researches were carried out to determine the action of this newer anthelmintic on the parasites of various domestic animals and on the hosts themselves.³ The results of this work indicated that the drug was effective against not only the hookworms and roundworms of dogs but also against these parasites in cats and black-silver foxes, the roundworms of swine and chickens and the stomach worms of sheep. It was found to be comparatively free from toxic effects when given in therapeutic doses.

The results embodied in this report are those obtained following the further use of this drug in various domestic animals and poultry.

INFLUENCE OF FATTY DIET ON ACTION OF TETRACHLORETHYLENE

Davis⁴ found that fats, oils, and cream were contraindicated in the use of carbon tetrachlorid as an anthelmintic, on account of being conducive to a maximal liver injury. Hall and Shillinger¹ thought that a high carbohydrate diet might be protective when tetrachlorethylene was administered to dogs, although the author found in previous work^{2,3} that a diet of mother's milk or whole cow's milk did not apparently affect the efficacy or toxicity of the drug when given to puppies.

In order that the influence of fat on the action of tetrachlorethylene might be determined, two dogs were fed uncooked beef suet (table I) some five hours before being dosed. Three hours after the administration of a therapeutic dose, the dogs were again fed beef suet, which made up their entire diet for the en-

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suing 24 hours. One dog (No. 3) vomited several times soon after the suet was eaten but did not vomit after the tetrachlorethylene had been given. No unusual symptoms were noted during the four days they were under observation. On autopsy the internal organs of both appeared normal. Neither dog had passed any worms, dog 3 having 12 live roundworms and 8 tapeworms in the intestines on autopsy. In the other (dog 4), 14 cecal worms were found.

TABLE I—*Tetrachlorethylene to dogs on fatty diet*

DOG	WT. (LBS.)	FAT OR OIL	AMT.	C ₂ Cl ₄	WORMS PASSED	WORMS POSTMORTEM		
						ROUND	TAPE	CECAL
3	15	Beef suet	½ lb.	2 cc	0	12	8	0
4	20	Beef suet	1 lb.	2 cc	0	0	0	14
1	15	Butter	½ lb.	2 cc	0	0	0	0
2	15	Butter	½ lb.	2 cc	0	1	0	0
5	12.5	Cod-liver oil	2 oz.	2 cc	0	6	10	0
6	17	Cod-liver oil	2 oz.	2 cc	0	1	2	0
7	14.5	Castor oil	¾ oz.	2 cc	0	1	75	0
8	15	Castor oil	1 oz.	2 cc	0	0	0	0

Two other dogs (1 and 2) were fed each one-half pound of butter with a small amount of bread. Five hours later each dog was given 2 cc of tetrachlorethylene. These animals were observed for five days, during which time no worms were seen in the feces. On autopsy no changes thought to have been due to tetrachlorethylene were seen. One roundworm was found in the small intestine of dog 2.

In order to determine the effect of tetrachlorethylene when given simultaneously with oils, two dogs were given each 2 cc of the anthelmintic in soluble elastic globules and 2 ounces of cod-liver oil as a drench. No symptoms of toxicity due to the drug were noted during the five days they were held nor were any worms passed by either. On autopsy the internal organs were apparently normal.

Six roundworms and 10 tapeworms were found in dog 5, while only one round and 2 tapeworms were seen in dog 6.

Two other dogs were given each 2 cc of tetrachlorethylene in the soluble elastic globules, together with ¾ ounce and 1 ounce of castor oil. No worms were passed by either dog. No symptoms of toxicity were noted nor were any abnormal changes seen on autopsy held on the fifth day after treatment. In the intestines

of dog 7 one roundworm and 75 tapeworms were found. None were found in dog 8.

It would seem from these results that, while the feeding of fats to dogs shortly before administration of tetrachlorethylene did not affect the toxicity of the drug, it did reduce the efficacy to zero. Similar results were obtained following the simultaneous administration of tetrachlorethylene and cod-liver oil or castor oil. For this reason it would seem advisable to eliminate fatty foods from the diet of dogs a short time prior to administration of tetrachlorethylene. Nor should oils such as cod-liver oil or castor oil be given simultaneously with the drug if full anthelmintic effect is to be desired.

EFFECT ON RABBITS

To determine the effect of tetrachlorethylene on rabbits ten of these animals weighing from 1310 to 2110 gms. each were given the drug in doses varying from 0.2 to 2.0 cc. Dosage at these rates produced a variation of 0.102 to 1.4 cc per kilogram of body weight (table II).

TABLE II—*Tetrachlorethylene to rabbits*

RABBIT	WT. (GMS.)	C ₂ Cl ₄ (CC)	RATE PER KG. (CC)	REMARKS
1	1960	0.2	0.102	Not affected
2	1540	0.4	0.246	Not affected
3	1640	0.6	0.361	Not affected
4	1420	0.8	0.563	Not affected
5	1500	1.0	0.66	Not affected
6	1310	1.2	0.91	Dead—48 hours
7	1310	1.4	1.4	Not affected
8	1400	1.6	1.13	Not affected
9	1600	1.8	1.12	Dead—6 days
10	2110	2.0	0.94	Dead—25 minutes

In all cases, the tetrachlorethylene was given in soluble elastic globules. Some difficulty was experienced in administering the 1-cc size especially. When a globule of this size was being given to rabbit 7, it was broken in the mouth. None of the contents was lost but the animal suffered convulsions almost immediately. Recovery took place in about five minutes. Similar accidents occurred when the larger capsules were being administered to rabbits 9 and 10. The ensuing convulsions shown by rabbit 9 persisted only about two minutes, after which the animal became normal. The convulsions in rabbit 10 persisted for 25 minutes, when the animal died. On autopsy the lungs were congested.

Rabbit 6 died 48 hours after being dosed, having shown diarrhea during this time. The liver showed little or no change. Rabbit 9 died after six days, having shown only diarrhea during the observation period. The liver was dark, very firm and showed some areas of coccidial infestation.

The remaining animals were destroyed on the sixteenth day. On autopsy rabbits 2 and 3 showed evidence of hepatic coccidiosis but no other changes. Ten bladder worms were found in the abdominal cavity of rabbit 1 but the organs were apparently normal. Three of these parasites were seen also in rabbit 7. The internal organs of rabbit 8 were apparently normal.

It would seem that in doses up to 0.66 cc per kilogram, tetrachlorethylene can be given to rabbits with comparative safety. Care must be used in administration, since there is danger of producing convulsions and death, if the capsule is broken in the mouth and the contents escape. These symptoms are no doubt a result of the mechanical irritation following inhalation of the drug. Larger doses may produce death after several days.

EFFECT ON CATS

When the previous work was carried on to determine the effect of tetrachlorethylene on cats, only a few of these animals were available. Later, thirteen cats, most of which were about one-half to three-fourths grown, were obtained and treated with tetrachlorethylene in doses from 0.4 to 0.6 cc each (table III).

TABLE III—*Tetrachlorethylene to cats*

CAT	WT. (KG.)	C ₂ Cl ₄ (CC)	DOSE PER KG. (CC)	WORMS PASSED (ROUND)	WORMS POSTMORTEM
378	1.59	0.4	0.25	0	0
121	1.59	0.4	0.25	0	0
231	1.13	0.4	0.35	2	0
122	2.04	0.6	0.28	1	0
131	2.04	0.6	0.28	0	0
315	1.25	0.4	0.32	0	0
330	1.13	0.4	0.35	0	0
240	1.36	0.4	0.29	0	0
123	1.02	0.4	0.33	5	0
124	1.70	0.4	0.23	0	0
369	1.70	0.4	0.23	2	0
191	1.93	0.6	0.31	0	0
142	2.61	0.6	0.26	0	0

The diet of these cats for two weeks prior to treatment consisted entirely of powdered whole milk dissolved in water. Food was withheld for about two hours after dosing, early in the morning.

No toxic symptoms were noted in any of the cats during the five days they were observed. However, cat 131 was found dead in its cage on the morning of the third day, although from all appearances it was normal the night before. On autopsy the liver and spleen were enlarged and congested. The kidneys were congested.

All the remaining cats were destroyed on the third day, autopsies showing nothing which was abnormal. No parasites of any sort were found in the intestines of any of these cats.

As concluded from former tests, the results of this experiment indicate that tetrachlorethylene at the rate of 0.2 cc per kilogram of body weight or fraction thereof is efficient against the roundworms of cats and is safe to administer.

TESTS ON CHICKENS

In earlier experiments it was found that tetrachlorethylene given to chickens of average size in doses of one cc was effective against roundworms and was not toxic.

Desiring further data as to the effectiveness of similar and smaller amounts of the drug, thirteen White Leghorn chickens, weighing from one to two pounds, were obtained. The first seven (table IV) were obtained from the local market, while the re-

TABLE IV—*Tetrachlorethylene in varying amounts to chickens*

CHICKENS	WT.	C ₂ Cl ₄ (cc)	WORMS PASSED (ROUND)	WORMS POSTMORTEM	
				ROUND	TAPE
8	1 lb. 5 oz.	0.1	0	64	0
26	1 lb.	0.2	0	30	0
6	1 lb.	0.4	0	3	35
20	1 lb. 5 oz.	0.6	0	13	0
47	1 lb. 3 oz.	0.8	0	0	0
36	1 lb. 5 oz.	1.0	0	0	36
9	15 oz.	1.0	0	0	0
72	1 lb. 12 oz.	1.0	0	0	0
73	1 lb. 12 oz.	1.0	0	0	0
75	2 lb.	1.0	0	2	5
76	2 lb.	1.0	0	0	0
77	1 lb. 8 oz.	1.0	2	0	122
450	1 lb. 12 oz.	1.0	1	0	0

mainder were purchased from a nearby poultryman. Only the general unthrifty condition of these chickens was taken as indication of possible parasitism.

They were placed on a diet of commercial scratch-feed, supplemented with roots, prior to treatment. In the case of the first group of seven, the fasting period before treatment was limited

to six hours, while in the latter group this time was extended to twenty hours.

Tetrachlorethylene in soluble elastic globules was administered to these chickens in doses varying from 0.1 cc each to 1.0 cc. All were fed three hours after being dosed, the food being the same as that given before treatment.

At no time during the three days these chickens were held for observation, were any toxic symptoms noted nor were any lesions seen on postmortem, which were thought to have been due to the effects of tetrachlorethylene.

The results of this experiment, summarized in table IV, indicate that tetrachlorethylene in doses of less than 1 cc to chickens is not effective in removing the roundworms present. A dose of 1 cc in most instances is effective against these parasites (birds 77 and 450). On the other hand, a dose of this size may have no effect whatever in some cases (bird 75). This apparent lack of anthelmintic action of certain drugs in some individuals has been noted by several authors.

However, from this and other work it was found that 1 cc of tetrachlorethylene to average-sized chickens would in most instances remove all or practically all of the roundworms present.

Further evidence that tetrachlorethylene has no effect on the tapeworms of chickens is shown by the number of these parasites found in the intestines of birds 6, 36, 75 and 77.

TETRACHLORETHYLENE TO CALVES

It had been found that tetrachlorethylene in doses up to 35 cc was not toxic to calves varying from 376 to 480 pounds in weight.

To obtain more data as to the effect of this anthelmintic on these animals, nineteen calves were used. They were from 1 to 1½ years of age and weighed from 350 to 550 pounds.

In preparation for treatment these animals were taken off of pasture which had been supplemented by mixed clover and timothy hay and removed to the stables where they were starved for about twelve hours. Tetrachlorethylene was then administered in doses of 15 to 38 cc. No symptoms of toxicity were noted during the three days they were held before slaughter nor were any apparent changes noted in the internal organs on post-mortem.

One calf weighing 400 pounds was dosed with 20 cc of tetrachlorethylene, followed in eight days by another dose of 35 cc. No toxic symptoms were seen nor were there any apparent

changes in the internal organs when the animal was slaughtered on the third day after administration of the last dose.

Unfortunately no data could be obtained as to the effectiveness of the drug against the parasites of cattle, since none were seen in the feces nor were any found postmortem.

From these experiments it is evident that tetrachlorethylene to calves, 1 to 1½ years of age, when given in doses up to 38 cc, is not toxic. Whether or not the non-toxicity of tetrachlorethylene for these animals was influenced by a diet high in calcium content, as is that of carbon tetrachlorid, is not known. Hall⁵ has recommended a diet of this sort for a week or more previous to administration of this latter drug in order to reduce the toxic effects. Further experiments are necessary to determine the effect of tetrachlorethylene on milch cows and on cattle on a diet deficient in calcium.

EFFECT OF LARGE DOSES ON DOGS

In previous tests with tetrachlorethylene to young dogs and puppies, it was seen that the drug given in doses up to twenty-five times the therapeutic dose (0.2 cc per kg. of body weight) caused no ill effects.

Desiring data as to the effect of these larger doses on adult dogs, five of these animals of varying size were used (table VI).

TABLE VI—Large doses of tetrachlorethylene to adult dogs

DOG	WEIGHT (KG.)	C ₂ Cl ₄ (CC)	DOSE PER KG. (CC)	REMARKS
1	6.59	5.0	0.70	Normal
2	11.30	10.0	1.13	Slightly dizzy
3	7.27	15.0	2.06	Normal
4	4.09	20.0	4.89	Normal
5	12.50	25.0	2.00	Slightly dizzy

In preparation for treatment, these dogs were fed as usual in the evening, after which all remaining food was removed. They were not fed again until two hours after being dosed. No purgatives were given before or after treatment.

Within an hour after treatment was administered, dog 2 became slightly dizzy. The others were apparently normal. The dogs were not seen again until next morning, when dog 2 was still slightly dizzy, dog 5 very much so, while the others were apparently unaffected. Dog 5 was still dizzy at 1:30 p.m., but returned to normal by evening except for a slight stiffness of the hind limbs which persisted until next morning. The remaining

dogs showed no clinical evidence of toxicity during the remaining period of observation.

It is interesting to note that dogs 3 and 4 were suffering from distemper at the time they were treated. The presence of this disease apparently had but little influence on the toxic effects of tetrachlorethylene.

No effort was made to determine the number of parasites passed by these dogs but none were found when autopsies were held on the sixth day after dosing.

The liver, spleen and kidneys of dog 1 were congested and firm. The capsule of the kidneys was slightly adherent. Some cloudy swelling also was noted. The intestines were apparently normal.

In dog 2 the liver was rather friable and congested. This latter condition also was present in the lungs, possibly due to beginning distemper. There was a slight amount of inflammation in the upper portion of the small intestine.

After dog 3 had been destroyed for autopsy, there were found slight areas of broncho-pneumonia in the right lung. The liver, kidneys and spleen were congested. The intestines appeared normal and contained no parasites.

The liver of dog 4 was slightly yellow in color, firm, and slightly mottled with small hemorrhagic areas. The gall-bladder was filled with bile of normal color and consistency. The spleen was firm and pale. Some cloudy swelling was seen in the kidneys. Broncho-pneumonia was present in both lungs. No parasites were found in the intestines, which appeared normal.

Autopsy of dog 5 showed nothing unusual except congestion of the liver, spleen and kidneys.

From this and previous experiments it is evident that dogs tolerate large doses of tetrachlorethylene exceptionally well. The hemorrhagic areas in the liver of dog 4 may possibly have been due to the effect of tetrachlorethylene but if so the lesions were not sufficiently severe to produce any clinical manifestations of toxicity. Occasional animals may show symptoms of dizziness or muscular incoordination, which symptoms usually pass off in a very short time leaving no apparent bad after effects.

EFFECT OF REPEATED DOSES TO SHEEP

Knowing the effectiveness of tetrachlorethylene against the stomach worms of sheep and realizing the necessity of frequent treatment of these animals for the removal of these parasites, especially when sheep are run on infested pastures, experiments

were carried out to determine the effect of this drug when given at intervals of approximately thirty days.

For this experiment ten aged ewes were purchased, all having been bred and were supposedly pregnant. In preparation for treatment all the ewes were ear-tagged, weighed and starved overnight. Five were then treated with 5 cc each of tetrachlorethylene in soluble elastic globules. The remaining five were left untreated as controls, and were used for this purpose during the entire experiment, which extended over a period of one year.

During the winter months the ewes were sheltered in a barn and were fed clover hay. They also had access to the pasture on which they were kept during the summer months. This pasture was not known to have been infested with ova of stomach worms since sheep had not been run on it for some years.

Once each month all animals were fasted overnight, weighed, and the five first treated were given each 5 cc of tetrachlorethylene.

Unfortunately all the ewes used were not pregnant at the beginning of the experiment, since only three (one treated and two controls) gave birth to lambs. The lamb from the one treated ewe was lost because of chilling soon after birth. One ewe gave birth to twins, which were later used as controls. The remaining lamb was first treated with 5 cc of tetrachlorethylene when about $3\frac{1}{2}$ months of age. Treatment with this amount was repeated at 30-day intervals until the end of the experiment.

Treatment of these sheep at these stated intervals was followed by no evidence that the drug was toxic, even to the one animal which was pregnant.

At the end of the year all the sheep, both ewes and lambs, were slaughtered. Nothing was seen in the internal organs which indicated any harmful results had followed the repeated use of tetrachlorethylene. It was noted, however, that the untreated controls were rather heavily infested with nodular worms, whereas the treated animals were only very slightly infested with these parasites. No stomach worms were seen in any of the animals of either group.

When the experiment was terminated, it was found that the average gain in weight of the untreated ewes (not including control lambs) had been 6 pounds per head for the entire period. The gain in weight in the treated animals averaged 8.6 pounds per head during this time. While the gain of the treated over that of the untreated sheep was not great, yet it indicates the possibility

of obtaining greater gains in weight when animals are kept as free as possible from internal parasites. Greater gains might have been made by the animals of both groups had a supplementary ration of grain been fed. On account of the wide differences in their ages, little or nothing could be determined as to the effect of treatment on the gains in weight of the lambs.

The experiment did show, however, that tetrachlorethylene could be given to sheep or lambs in doses of 5 cc at intervals of thirty days without harmful effects. Used in this manner the drug should prove very effective in the control of stomach worms in sheep and would at the same time aid in reducing the amount of infestation with nodular worms.

SUMMARY

When tetrachlorethylene is given to dogs whose diet has consisted largely of fat for a short time before treatment or when the drug is given simultaneously with cod-liver oil or castor oil, its efficacy is reduced to a minimum.

Tetrachlorethylene can be administered to rabbits in doses up to 0.66 cc per kilogram of body weight. Greater amounts than this in most cases appear to be toxic, death following soon after administration or within a few days.

A dose of 0.2 cc per kilogram of body weight or fraction thereof is sufficient to remove the roundworms from cats. Doses of this size are tolerated exceptionally well by these animals.

The roundworms present in chickens of average size are, in most cases, removed by tetrachlorethylene in doses of 1 cc. Smaller amounts do not seem effective in removing these parasites.

Calves, 1 to 1½ years of age, tolerated tetrachlorethylene, in doses up to 38 cc, exceptionally well. A dose of 20 cc, followed in one week by 35 cc, was not toxic to one calf. It was not known whether or not the diet rich in calcium, obtained by including relatively large amounts of clover hay, caused the apparent lack of toxicity of the drug to these animals.

Adult dogs tolerate large doses of tetrachlorethylene very well. An occasional animal may show symptoms of dizziness which are usually transitory, passing off within a few hours and leaving no apparent bad after-effects. The dogs suffering from distemper did not seem to be any more susceptible to the action of the drug than did those which were normal. In most cases, however, when dogs are suffering from distemper, it would no doubt be ad-

visible to postpone anthelmintic treatment with tetrachlorethylene until recovery had taken place.

Tetrachlorethylene given to sheep or lambs in doses of 5 cc was not toxic. No apparent harmful effects were noted following the repeated use of this drug in the pregnant animals.

CONCLUSIONS

1. Fats should be eliminated from the diet of dogs a few days prior to administration of tetrachlorethylene if full anthelmintic effect of the drug is to be obtained. Likewise the drug should not be given simultaneously with cod-liver oil or castor oil.

2. Tetrachlorethylene in doses exceeding 0.66 cc per kilogram of body weight is toxic for rabbits in most instances, and may cause death shortly after administration or within a few days. Smaller doses seem to be well tolerated. Care must be used in the administration of the drug in order that none escapes into the mouth. If such an accident should occur, suffocation, due to the mechanical action of the liquid, may occur, recovery or death following, dependent on the amount liberated.

3. Two-tenths of one cc of tetrachlorethylene per kilogram of body weight or fraction thereof is sufficient to remove the roundworms from cats. No toxic effects should be noted following the use of the drug in doses of this size.

4. One cc of tetrachlorethylene, in most cases, is sufficient to remove the roundworms from chickens of average size. Smaller amounts are not efficient in removing these parasites. The drug has no effect on tapeworms of chickens.

5. Calves, 1 to 1½ years old, tolerate tetrachlorethylene in doses up to 38 cc without displaying either clinical symptoms or postmortem lesions of toxicity.

6. Large doses of tetrachlorethylene can be given to adult dogs without the production of toxic symptoms other than those of dizziness in an occasional animal. These are transitory and usually disappear within a few hours. Dogs suffering from distemper tolerated the drug as well as the normal ones. Under ordinary conditions, however, it would seem advisable to postpone anthelmintic treatment with tetrachlorethylene until recovery from distemper has taken place.

7. Tetrachlorethylene can be given to sheep in doses of 5 cc and repeated at intervals of 30 days without harmful effects. Given at these intervals the drug should be effective in controlling stomach worms in sheep run on infested pastures. Used in this

manner, it should also be effective in reducing infestation with nodular worms. No harmful effects should follow the use of the drug in pregnant ewes providing proper precautions are observed when they are being handled.

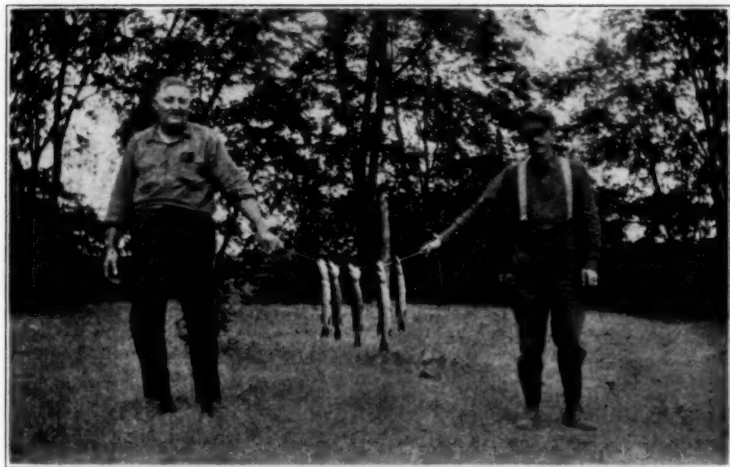
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Missouri Valley Meeting

Secretary Steel announces a very interesting program for the meeting of the Missouri Valley Veterinary Association, to be held at Omaha, Nebr., July 8-9-10. The first two days will be given over to business and the reading of papers. On the third day a clinic will be held at the stock-yards. This will be divided into four sections: swine, poultry, small animals and cattle. Among those who will contribute to the program is Dr. T. E. Munce, Harrisburg, Pa., president of the American Veterinary Medical Association.

Michigan is the summer playground of America—Detroit is the gateway.

**COME TO MICHIGAN TO FISH**

Dr. Donald B. Palmer (on the right), of Minneapolis, Minn., caught these five great northern pike (*Esox lucius*) about twenty miles from Muskegon, Mich., in the month of August. Members who attended the Minneapolis convention will recognize Dr. Palmer as the chairman of the Sub-Committee on Publicity.

CORRELATION OF THE RAPID AND THE SLOW AGGLUTINATION TESTS FOR BANG'S ABORTION DISEASE OF CATTLE

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The development of the rapid agglutination test for the diagnosis of Bang's abortion disease, by Huddleson and his co-workers, constitutes, in the opinion of the writers, an important contribution to veterinary medicine. The routine testing of cattle for Bang's abortion disease, as a part of a control program or for the purpose of shipping cattle into states requiring a negative serum test for entry, is showing a marked growth. If the future developments in this disease require the testing of cattle on a scale comparable with that of tuberculosis, and abortion-free accredited herds become the ultimate goal, as is now the case in a few states, the laboratories now in existence will be seriously embarrassed in handling the great volume of testing work. Either the laboratories will be required to increase greatly their facilities for handling this work or the test must be conducted by the veterinarians in the field. We believe practitioners should be accredited for testing cattle for Bang's abortion disease, just as they are accredited for testing herds for tuberculosis. In many herds it will be found practical for the local practitioner to conduct tuberculosis and abortion tests simultaneously.

With the tests formerly in use, namely, the complement-fixation and the slow (test-tube) agglutination, the general practitioner, in most instances, would not be expected to participate in the work of testing other than collecting blood samples. He would prefer relegating the work of making the test to a laboratory worker. The rapid macroscopic test now places in the hands of the practitioner an accurate test that is comparatively simple in technic. It is conducted with only a few pieces of apparatus, requires no incubator, extensive pipetting or test-tube arrangement, and the comparatively simple equipment may be easily set up and the test conducted in any office.

Comparative studies of the rapid or plate method with the slow or test-tube method reported in the literature to date show only

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slight disagreement. Huddleson and Carlson¹ are convinced that the rapid method is just as accurate as the present slow-agglutination method for the serum diagnosis of Bang's abortion disease in cattle. They report 100 per cent agreement for 2000 samples of bovine sera where clumping was complete or incomplete in 0.04 cc or less of serum. Lienhardt and Kitselman² found a disagreement of approximately one per cent, or 0.5 per cent for each method. They state:

in this laboratory the short method has proven to be highly satisfactory and because of the ease with which the test can be performed and the speed with which a diagnosis can be made is to be preferred and will become a permanent diagnostic method in the testing of all routine bovine sera for Bang's abortion disease.

Although the time factor in conducting the agglutination test is an important one, the chief advantage which the rapid method possesses over the slow method is found in the simplification of the technic, especially in laboratory equipment.

METHODS

The method described by Huddleson and Abell³ for conducting the rapid macroscopic agglutination test was employed for the rapid test. Their instructions for the preparation and standardization of the antigen, the apparatus and the technic of conducting the test were carefully followed in detail, with the exception of using a dark-field illumination box to produce a black background. This apparatus, while of unquestionable value for making readings, may be dispensed with if the glass plate is placed upon an ordinary laboratory table with a black background.

Leinhardt and Kitselman encountered minor objections to the Huddleson and Abell technic, namely, the smallness of the ruled squares on the glass plate, these workers preferring larger ruled squares. They recommend sterile pipettes for mixing the antigen and the serum rather than toothpicks. These objections did not occur to the writers. No difficulties were experienced with the test fluid overrunning the squares when the plate was slowly tilted back and forth. Toothpicks for mixing the antigen and serum proved satisfactory. They are inexpensive and save considerable time as they are destroyed after being used, whereas, pipettes require considerable time for cleaning and sterilizing. It is to be expected, however, that different workers will prefer minor variations in technic.

The plate employed in this study was arranged for handling 24 sera at one time. We agree with Huddleson and Abell, that the

best results are obtained by testing only ten or twelve sera at a time.

In conducting the rapid test it is important that the plate serum and antigen should be kept at room temperature or slightly above, otherwise the reactions will be delayed. If the antigen and sera are stored in the ice-box they should be brought to room temperature before the test is started, and the plate should be warmed to about body temperature. When these precautions are followed, reactions occur almost immediately and any reaction occurring later than five minutes may be disregarded. Occasionally delayed reactions do occur, but when the serum giving such a reaction is tested by the slow method it is usually negative.

In conducting the slow (test-tube) test, the writers employed six dilutions. A preliminary or stock dilution of 1 to 10 is made first by adding 0.4 cc of serum to 3.6 cc of carbolated physiological salt solution. From this stock solution of the serum the following amounts were added to one cc of the antigen: 0.4, 0.2, 0.1, 0.05, 0.02, and 0.01 cc, resulting in dilutions of 1-35, 1-60, 1-110, 1-210, 1-510 and 1-1010.

PREPARATION OF ANTIGEN

The antigen for the slow test was prepared by removing the growth with carbolated physiological salt solution and standardizing to compare with tube one of the McFarland nephelometer. After standardizing the antigen, it was heated in a water bath to 100° C. for ten minutes and filtered through absorbent cotton to remove the coagulated material. The heating and filtering of the antigen increases its sensitivity to the action of specific agglutinins. The antigens for both the rapid and slow tests were prepared from several strains of *A. abortus*, the agglutination ability of which had been determined previously by the slow macroscopic method. The organisms for both antigens were grown upon the same kind of media contained in Blake bottles. Sufficient quantities of the two antigens were prepared at the same time for conducting the tests reported in this series.

When serum samples were received for testing, the rapid and slow tests were started simultaneously. From a sterile pipette containing the serum to be tested 0.4 cc was delivered into a test-tube containing 3.6 cc of carbolated salt solution. This constituted the 1-10 or stock dilution previously described for the slow (test-tube) test. From the same pipette 0.08, 0.04, 0.02, 0.01

and 0.004 cc of the serum were delivered to the appropriate squares of the plate used in conducting the rapid test. While one investigator completed the rapid test, the other completed the slow test. The samples were run in batches of twelve or less and the operators completed their technic in approximately the same length of time. The reading of the rapid test was completed at once (within five minutes), whereas, the test-tubes containing the serum dilutions and antigen of the slow test were incubated for 48 hours, followed by standing at room temperature for an additional 24 hours before reading.

RESULTS

Sixteen lots of sera, comprising 609 samples, were tested simultaneously by the rapid and slow macroscopic methods for Bang's abortion disease. The rapid method gave 94 reactors, whereas, the slow method gave 83 reactors. A study of the individual lots, as shown in table I, reveals that in exactly one-half

TABLE I—Comparison of rapid and slow agglutination tests

LOT	SAMPLES	POSITIVE		NEGATIVE		AGREEMENT (%)
		RAPID	SLOW	RAPID	SLOW	
1	28	0	0	28	28	100.00
2	62	16	17	46	45	98.39
3	51	7	5	44	46	96.08
4	62	8	6	54	56	96.78
5	62	3	1	59	61	96.78
6	5	1	1	4	4	100.00
7	25	2	1	23	24	96.00
8	27	0	0	27	27	100.00
9	18	1	1	17	17	100.00
10	30	5	5	25	25	100.00
11	29	10	11	19	18	96.55
12	30	4	4	26	26	100.00
13	47	6	6	41	41	100.00
14	97	28	23	69	74	94.85
15	6	3	2	3	4	83.34
16	30	0	0	30	30	100.00
Totals	609	94	83	515	526	Mean 97.539

of the group (8 lots) the agreement was in every case 100 per cent. Eight of the lots show disagreement in the two methods. All of the lots show a high percentage of agreement except lot 15. In this lot the rapid test gave three positive reactions, whereas, the slow test gave two positive reactions. This is a disagreement of one, but when figured upon a percentage basis for a lot containing only six samples it results in an agreement of only 83.34 per cent.

Using total number of samples for frequency values and percentage of agreement as class values, there results a mean of 97.539, S. D. 2.37, and P. E. m of .6529. This gives us odds of 64.79:1. Since any determination giving odds greater than 30:1 is significant, the above method must be significant. With these odds of 64.79:1 there is only 1.52 chances in 100 that it is not correct.⁴

SUMMARY AND CONCLUSIONS

Six hundred nine samples of bovine sera were tested simultaneously by the rapid and slow macroscopic agglutination tests for Bang's abortion disease. The method described by Huddleson and Abell was employed for the rapid test. The slow test was conducted with the usual six dilutions of serum, ranging from 1-35 to 1-1010.

The two antigens were prepared at the same time from several strains of *A. abortus*. The antigen for the rapid test was prepared and standardized after the method of Huddleson and Abell. The antigen for the slow test was standardized to compare with tube one of the McFarland nephelometer. Sufficient quantities of the two antigens were prepared for conducting all of the tests reported in this series.

The rapid test gave a total of 94 positive reactions, whereas the slow test gave 83 positive reactions. The samples consisted of sixteen lots varying in number from 5 to 97. In eight, or one-half of the lots, the results in the two tests agreed 100 per cent. Six of the lots gave a larger number of positive reactors to the rapid test than to the slow test. Two lots gave more positive reactors to the slow test than to the rapid test.

Using the total number of samples for frequency values and the percentage of agreement as class values, there resulted a mean of 97.539, a S. D. of 2.37 and a P. E. m of .6529.

It appears from this study that the rapid agglutination test is just as accurate as the slow agglutination test for the serum diagnosis of Bang's abortion disease.

The rapid test is easily performed, requires no expensive laboratory equipment and should become a permanent diagnostic method in the routine testing of bovine sera for Bang's abortion disease.

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RABIES CONTROL IN CHICAGO

By HENRY C. BECKER

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A great many diseases of animals are primarily of great economic importance, and are the cause of enormous financial loss. This is particularly true of diseases affecting meat and food animals. Certain animal diseases are of much greater significance, however, in that they are transmissible from animal to man. In the wide range of scientific research, discovery and experiment in the study of animal diseases, there is perhaps none more vital and more important than the disease known as rabies. While rabies is one of the oldest known diseases, there is probably none about which people generally have so many erroneous opinions and ideas as that regarding the cause, nature and transmissibility of this disease.

PUBLIC HIGHLY OPINIONATED

In spite of the proved infectious character of rabies, it is still believed by many that it is caused by great heat, thirst, nervous excitement, too high feeding, and numerous other causes. Many also believe that rabies does not occur in winter, but is confined solely to the summer months. It is because of this misunderstanding the public have generally regarding the disease, that makes it difficult to secure public cooperation in the endeavor toward eradication. In the presentation of this paper on rabies, it is recognized that we as veterinarians are thoroughly familiar with the cause, symptoms and transmission of this disease. I therefore deem it unnecessary to dwell upon these phases of the subject.

The status of rabies control in the city of Chicago during the past year has assumed the proportions of an epidemic. Rabies in Chicago had been at a low point for a number of years. During the entire year of 1926 there were examined in our laboratory only 157 dogs' heads for rabies, of which 17 (11 per cent) were found to be positive. Beginning early in the year 1927, the disease gradually increased and by the end of the year it was prevalent to an alarming extent. During 1927, 344 heads were examined of which 146 (42 per cent) were found to be positive. In the

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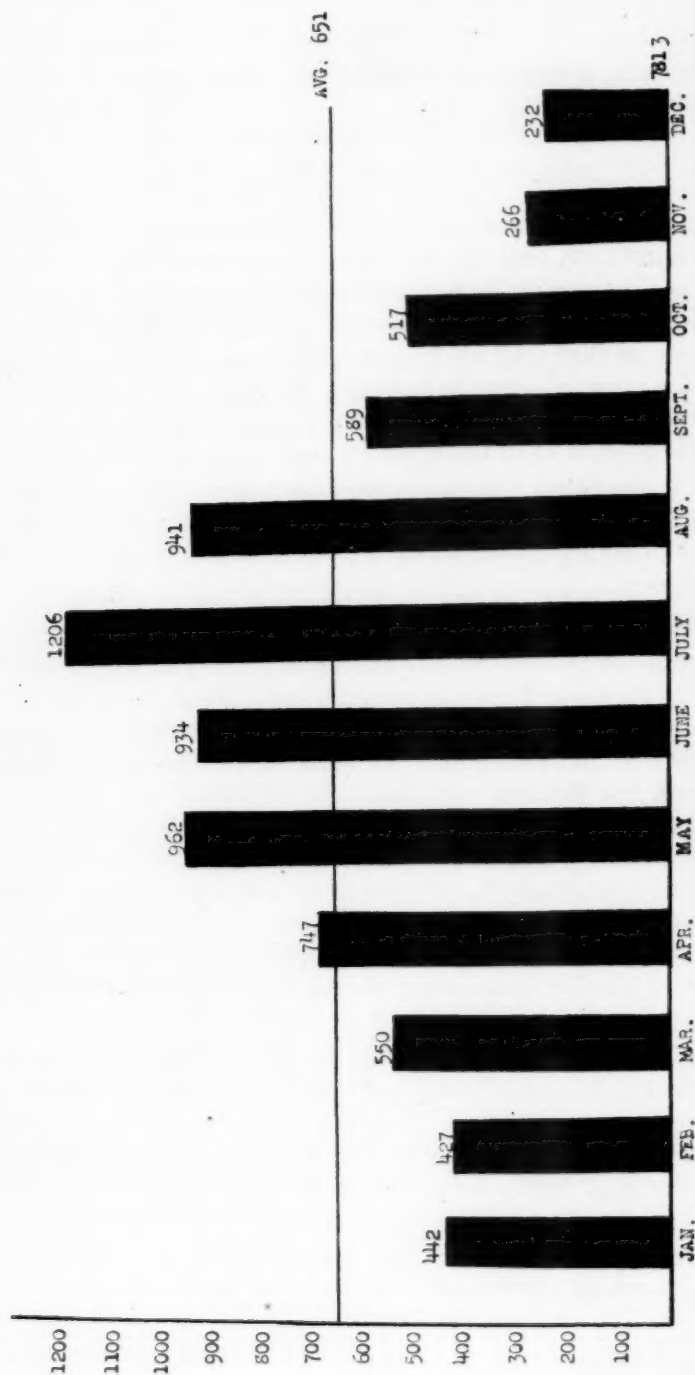


FIG. 1. Graph of dog-bite cases for the year 1928, Chicago Department of Health

period covered by the last five months of 1927, there occurred in Chicago *seven human deaths* as the result of dog bites. This was a greater number of human deaths from rabies than has occurred in Chicago for the entire period from 1915 to 1926 inclusive.

CAMPAIGN AGAINST RABIES

Realizing the seriousness of the situation, the Commissioner of Health, Dr. Arnold H. Kegel, began an active campaign on December 1, 1927, toward the eradication of rabies in the city of Chicago. This campaign in general included the following:

1. Conferences were held with representative veterinarians throughout the city, for the purpose of securing their cooperation, the exchange of ideas, and the laying out of a uniform line of procedure to be followed in the campaign.

2. Educational propaganda for the public through the daily press and preparation of special bulletins for general distribution, designed with a three-fold purpose.

First, as a measure of warning or caution to the citizens of the city of Chicago against the impending danger of rabies, which was widely prevalent among dogs in the city. Second, as a source of simple information on rabies which everyone should know, and which might serve as a safeguard against the disease. Third, as an appeal for the cooperation of every citizen, more especially the dog-owners, in obtaining strict compliance with the law governing the keeping of dogs in the city and in exerting a determined effort toward eradicating this dreaded disease in animals in the shortest possible time, thus protecting and saving human life.

3. Conferences with health officers of cities surrounding Chicago with a view to enforcing uniform regulations.

4. Enlisting the support of the Department of Police, who, in Chicago, are charged with the enforcement of the ordinances relating to dogs.

5. Cooperation of the state authorities for the enforcement of regulations in and out of Chicago.

6. Requiring the medical profession to report all dog-bite cases and the investigation of them by the Department of Health. All dogs reported to have bitten persons were required to be placed under observation either at the dog pound or placed in the custody of a licensed veterinarian for a period of not less than two weeks. Arrest slips were issued upon owners in all cases where they refused to give up the dogs. (I might say that this method of securing biting dogs proved very effective.)

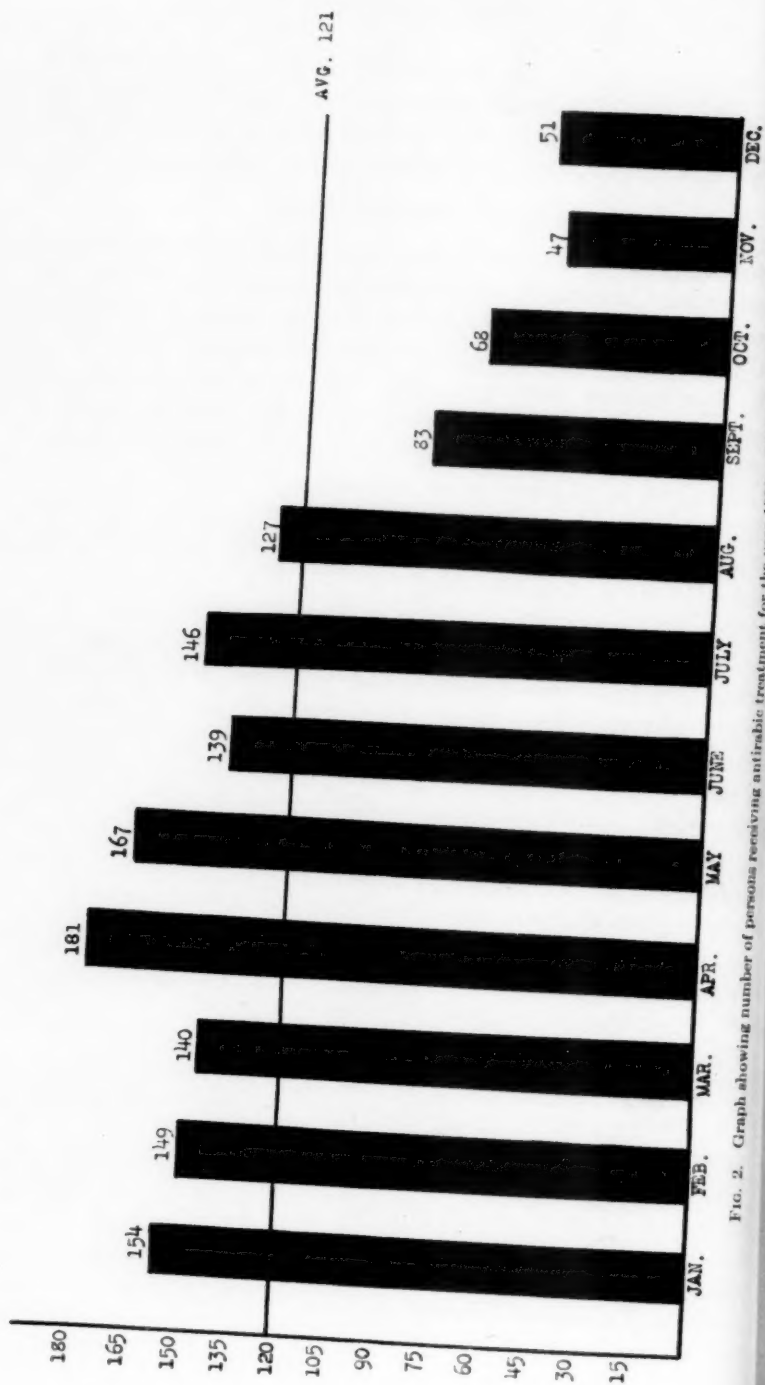


FIG. 2. Graph showing number of persons receiving antirabic treatment for the year 1928. Chicago Department of Health.

7. Providing free of cost the antirabic treatment for persons bitten by rabid animals in cases where they were financially unable to pay for same.

8. Advocating and recommending the immunization of all dogs against rabies as an added measure of protection.

9. The issuance of a proclamation requiring all strays picked up on the streets, and biting dogs impounded for observation, to be immunized before release by a qualified veterinarian, and that stray dogs be held only 24 hours before being destroyed instead of holding them 3 days, as is required by city ordinance.

On January 18, 1928, the proclamation referred to was issued by the Commissioner of Health worded as follows:

Department of Health

City of Chicago

Proclamation

On account of the widespread prevalence of rabies among the stray dogs of the city of Chicago, divers persons have been bitten by dogs affected with this disease. Eight deaths have already occurred among persons thus bitten, due to the failure to give the Pasteur preventive treatment promptly. There is also evidence that rabies is being transmitted to rats and other animals in the city.

Within recent years an increase has occurred in the number of unlicensed stray dogs in the city, so that the Dog Pound is at present unable to accommodate the dogs that are being caught at large on the streets, if such animals are held three days, as required by the city ordinance.

Because of the large proportion of dogs infected, great danger of the spread of rabies exists among the dogs caught and impounded.

In view of these facts, I hereby declare that an emergency exists, and, in accordance with the authority vested in me by Section 1781 of the Revised Municipal Code of 1922, I make the following ruling, to be effective this date:

No unlicensed dog shall be required to be held longer than twenty-four hours (24) in any dog pound, dog refuge, place or establishment where dogs are impounded, harbored or kept for the purpose of destruction or reclamation; provided that this shall not apply to biting dogs, held in accordance with Section 1059 of the aforesaid Municipal Code.

No dog shall be released, given out, sold or delivered from any dog pound, dog refuge, place or establishment where dogs are impounded, harbored or kept for the purpose of destruction or reclamation, unless said dog is first placed in care and custody of a veterinary surgeon, who will guarantee to immunize such dog against rabies and hold it in quarantine until it has been completely immunized in an approved manner. Such immunization and quarantine shall also be required for biting dogs, released from the City Dog Pound.

All dog owners are warned to keep their dogs confined on the premises, away from other dogs, or muzzled and on leash while on the street or public places, for a period of sixty days, during which time all dogs at large will be caught and impounded.

(By) ARNOLD H. KEGEL, M. D.
Commissioner of Health,
City of Chicago.

January 18, 1929.

COOK COUNTY UNDER STATE QUARANTINE

About the time this proclamation was issued, the State Department of Agriculture declared Cook County under quarantine.

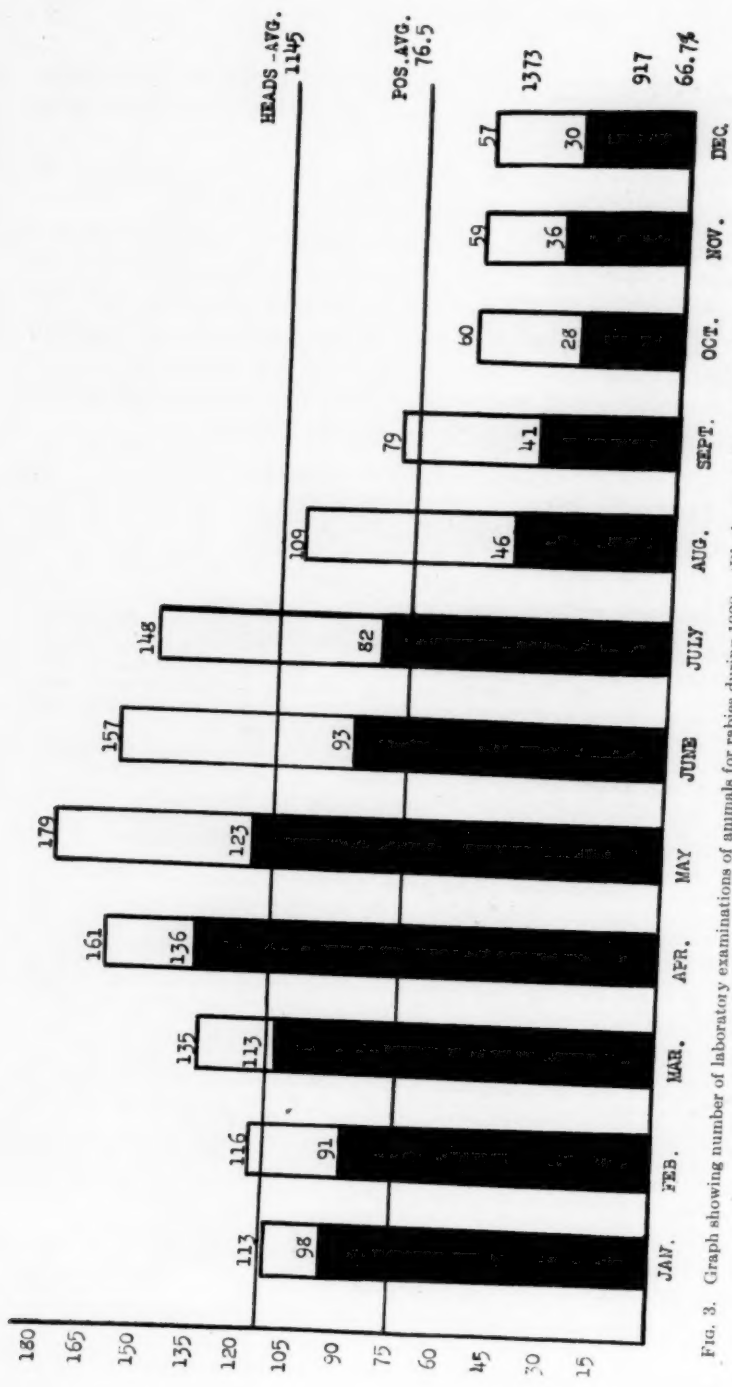


Fig. 3. Graph showing number of laboratory examinations of animals for rabies during 1928. (Black area indicates positive heads; white area, negative heads.) Chicago Department of Health

In effect this state embargo prohibited any dogs being brought into or taken out of Cook County without first being given a physical examination and also vaccinated against rabies by an assistant state veterinarian. This quarantine order without question had the desired and decided effect of retarding the movement of dogs, thus adding greatly to the prevention of disseminating the disease. Within recent years much has been accomplished by scientists in the endeavor to immunize dogs successfully against rabies by preventive inoculation. The matter of requiring the compulsory vaccination of all dogs in the city of Chicago was given careful and thorough consideration. The literature covering the subject was carefully reviewed, and the department communicated with many large cities throughout the United States where the vaccination method had been tried out.

There seems to be a division of opinion, both on the part of the investigators and the enforcement officials of the different cities, as to the value and reliability of the antirabic vaccination of dogs. While vaccination is compulsory in many cities it appears that careful follow-up observations have been lacking, rendering it impossible to measure the value of vaccination as a factor in controlling the disease. While the immunization method of prevention is not absolute, the degree of success attained experimentally, both in this country and in Japan, in producing an immunizing vaccine against rabies is most encouraging. *It should be strongly urged that this method of protection be used, but only as an added precaution, without relaxing other control methods.*

ELIMINATE OWNERLESS DOGS

In Chicago the success of rabies eradication is greatly dependent upon ridding the city of stray and ownerless dogs, of which there were and are still many thousands. For over a year an accurate tabulation has been kept by days, of the four major activities which were used as an index to show the progress of the eradication campaign. This included: First, dog-bite cases reported to the Department of Health, i. e., persons bitten. Second, number of persons given antirabic treatment by the Department of Health. Third, number of animal heads examined for laboratory diagnosis, and the number found positive. Fourth, number of stray and biting dogs impounded and destroyed.

I have prepared four charts showing by months the four activities just enumerated for 1928. During the year 1928 there

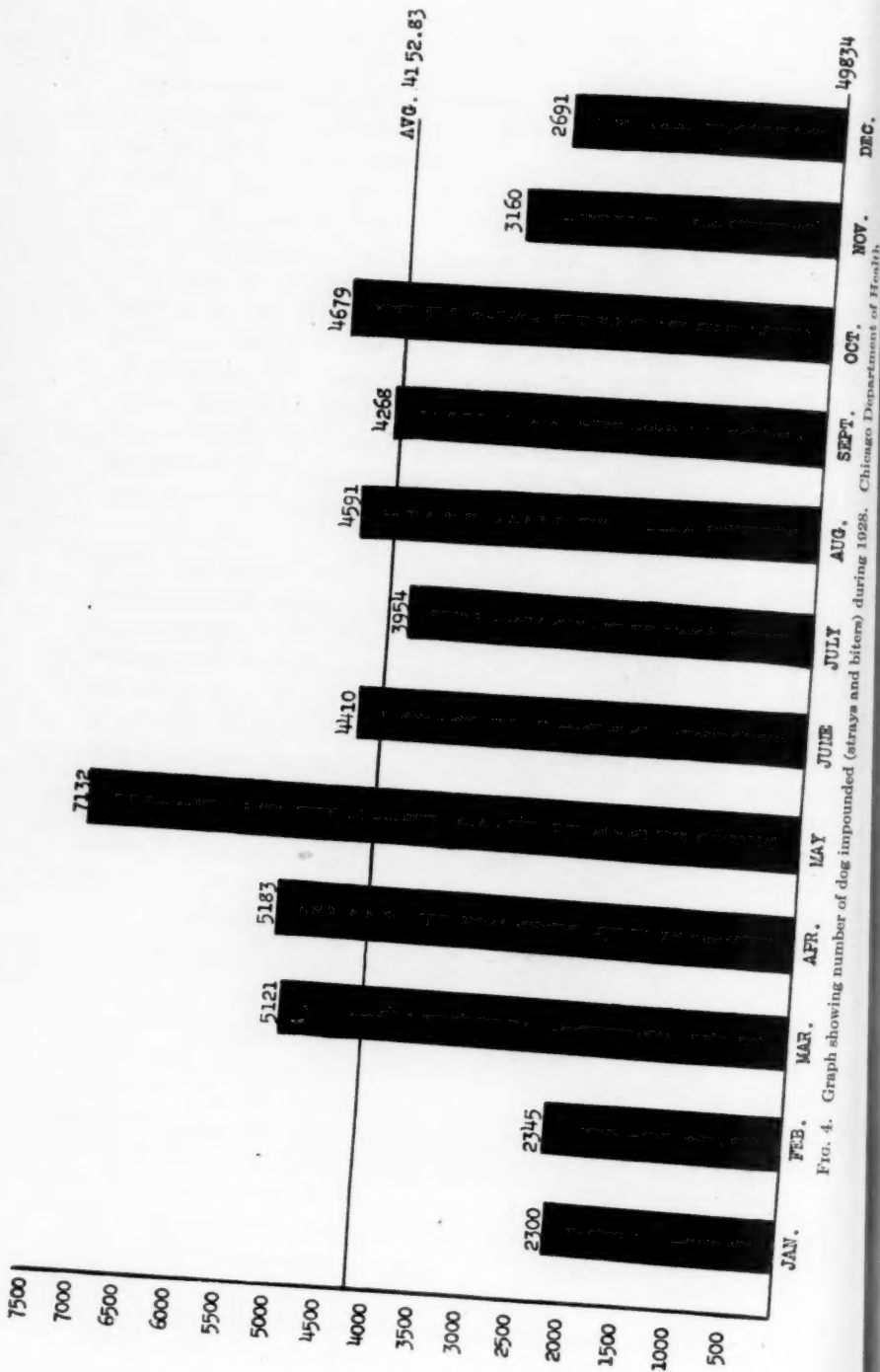


FIG. 4. Graph showing number of dog impounded (strays and biter) during 1928, Chicago Department of Health.

were reported to the Department of Health 7813 persons bitten by suspected rabid dogs. This is shown by months in chart 1. The greatest number of persons reported bitten was in the month of July, when 1206 bites were reported. There has been a sharp reduction in the cases reported the last five months of the year and in December there were 232 bite cases reported.

Chart 2 shows by months the number of persons given the antirabic treatment. During the year 1928, 1452 persons were treated. The greatest number in any one month was in April, when 181 persons were treated. There has been a gradual reduction since April, and in December, 51 persons were treated.

Chart 3 gives by months the number of heads examined for laboratory diagnosis. The numbers at the top show the number of heads examined, while the numbers below give the number and percentage positive. During the year 1928, 1373 animal heads were examined, of which 917 (66.7 per cent) were found to show positive evidence of rabies.

In January, 86.7 per cent of the heads examined were positive. There has been a gradual reduction in both the number of heads examined and the percentage positive since May, and in December, 57 heads were examined, with 30 (52.6 per cent) positive.

Chart 4 shows, by months, the dogs impounded and destroyed. During the year 1928, there were picked up and destroyed 49,834 dogs, or an average of over 4,000 dogs per month. The highest month was in May, when 7132 dogs were impounded. In December there were 2691 dogs impounded.

It is regretted that there occurred, during 1928, six human deaths from rabies. These, added to the seven deaths in the latter part of 1927, make a total of thirteen deaths from rabies in practically one year. Yet, using the figures as an index, we feel that we have made considerable progress in the endeavor toward eradication. It is realized, however, that a good start has but been made and our best efforts are challenged in completely eradicating the disease from our city. Acknowledgment is made of the sincere and hearty cooperation extended the Commissioner and the Department of Health by the members of the veterinary profession in Chicago during our eradication campaign.

***In the year of our Lord, 1929, and of the A. V. M. A., 66,
you can combine business and a pleasurable vacation.
Detroit, August 13-16.***

CLINICAL AND CASE REPORTS

(Practitioners and others are invited to contribute to this department reports of unusual and interesting cases which may be helpful to others in the profession.)

A NOTE ON THE USE OF MUSTARD IN THE TREATMENT OF SHEEP FOR PARASITES

By WILLARD H. WRIGHT, Washington, D. C.
Zoological Division,
Bureau of Animal Industry, U. S. Department of Agriculture

INTRODUCTION

Some time ago a manufacturer of mustard supplied the Zoological Division with a quantity of its product for test in the treatment of the sheep stomach worm, *Haemonchus contortus*. The manufacturer stated that there was a growing demand for this product for the above purpose throughout parts of the British Empire.

A search of the literature reveals few references to the use of mustard as an anthelmintic. We have an unverified reference to a paper by Shaw (1919) to the effect that mustard is used in West Virginia in the proportion of 1 ounce of ground mustard and 1 ounce of copper sulphate to 2 quarts of water, the dose of the solution being 3 ounces for full-grown sheep.

Branford (1920),¹ working in the Punjab, India, recommends the mustard-copper sulphate treatment for stomach worms in sheep. He makes up 4 ounces of copper sulphate and 4 ounces of mustard to 3 gallons of water and uses a dose of 2 to 4 ounces of this for lambs, and makes up 4 ounces of copper sulphate and 4 ounces of mustard in 2½ gallons of water and uses a dose of 4 ounces of this for sheep.

TESTS

In view of the reported widespread use of mustard, it seems advisable to carry out tests to determine its anthelmintic value. In order to judge whether mustard of itself possessed any such action, the preparation was first tested alone.

Sheep 1 was given 8 grams of mustard in a capsule. All feces passed for the next four days were collected and washed through

¹Received for publication, May 10, 1920.

a series of screens. No worms were recovered. The animal was slaughtered on the fourth day and the following parasites collected postmortem: 1 *Haemonchus contortus*, 7 *Monodontus trigonocephalus*, 1 *Esophagostomum columbianum* and 25 small trichostrongyles. Mustard in this test apparently failed to have any action on the worms present.

Sheep 2 was given 8 grams of mustard in a capsule after being fasted 12 hours. No worms were passed for 2 days; the third day, 6 *Monodontus trigonocephalus* were passed; the fourth day, no worms. The animal was killed the fourth day after dosing and the following parasites recovered postmortem: 124 *Haemonchus contortus* alive in the abomasum, 2 dead in the colon; 55 *Monodontus trigonocephalus*, 32 *Esophagostomum columbianum* and 36 *Nematodirus* spp. Considering the two stomach worms found in the colon and the 6 hookworms passed as having been destroyed by the treatment, the indicated efficacy of mustard in this test was 1.6 per cent against stomach worms and 10 per cent against hookworms.

A third test was carried out in which the mustard was combined with a copper sulphate solution in accordance with the directions on the container furnished by the manufacturer of the mustard. These directions call for a 1 per cent solution of copper sulphate, containing 1 per cent of mustard, with the dose stated as 4 ounces for adult sheep.

Sheep 3 was dosed with 4 ounces of the 1 per cent copper sulphate-mustard solution, after being fasted 12 hours. No worms passed the first day; the second day, 3 *Esophagostomum columbianum* were passed; the third and fourth days, no worms. The animal was killed on the fourth day and the following parasites were recovered postmortem: 700 *Haemonchus contortus*, 80 *Monodontus trigonocephalus*, 14 *Esophagostomum columbianum*, 35 *Nematodirus* spp. and 5 *Moniezia* spp. The indicated efficacy of the copper sulphate-mustard solution in this test was 17.6 per cent for nodular worms.

DISCUSSION

Subsequent to the tests, we learned that mustard is used extensively in the treatment of stomach worms of sheep in New South Wales and a communication was addressed to the Department of Agriculture of New South Wales requesting information on the treatment. In reply to this communication, Dr. Max Henry, Chief Veterinary Surgeon, writes as follows:

I have to inform you that this preparation is recommended by this Department and its use is increasing in Australia.

This Department advocates the use of mustard in conjunction with copper sulphate on the following grounds: Copper sulphate in solution has a distinctly astringent effect on the alimentary tract and in addition it arrests the action of enzymes and organized ferments. In order to overcome this action of copper sulphate the stomachic, carminative and stimulant action of mustard is enlisted.

In these tests mustard alone showed a slight efficacy against stomach worms and hookworms and in combination with copper sulphate, a slight efficacy against nodular worms. It is possible that its efficacy against stomach worms was greater than that indicated on the basis of worms passed. Hall and Shillinger (1923)² called attention to the fact that stomach worms killed by anthelmintics are probably digested or disintegrated in the intestinal tract of the sheep and that all worms destroyed cannot be recovered intact from the feces. However, the fact that copper sulphate and mustard left 700 stomach worms surviving the treatment does not look very promising. We would not care to conclude very much from this one test, but we note in passing that we have not had nearly so many survivors in our tests of copper sulphate alone.

It was noted in these tests that the one animal given copper sulphate-mustard solution apparently relished it and objected less to the taste of the combined solution than sheep usually do to the copper sulphate solution alone.

In tests which are now being conducted and which will be reported on later, sheep dosed weekly with a 1 per cent solution of copper sulphate over a period of several months have apparently suffered no deleterious effects from such frequent administration of this drug. Kammlade and McCulloch (1928)³ report tests with various anthelmintics in which the lambs receiving the copper sulphate solution made gains equal to or exceeding those of the lambs dosed with other anthelmintics and those of the lambs in the control lot. The experiments already reported from this Bureau on the copper sulphate treatment indicate that for the cases in which *Haemonchus contortus* is the cause of trouble, and other parasites are of little importance, the copper sulphate treatment is very effective and satisfactory. It would appear, therefore, that the repeated administration of the 1 per cent solution of copper sulphate alone is not detrimental to the health of the animals, and we find no evidence indicating that it has had effects which may be offset by the use of mustard. The mustard may add some desirable qualities to the solution; we have no

evidence on this point and would reserve judgment. If it makes the work of drenching easier, by increasing the palatability of the solution, this might be sufficient reason for using it.

SUMMARY

Mustard alone exhibited very little efficacy against stomach worms (*Haemonchus contortus*) and hookworms (*Monodontus trigonocephalus*) of sheep.

In one test a 1 per cent solution of copper sulphate and mustard removed a small percentage of the nodular worms present.

The inclusion of mustard in the 1 per cent solution of copper sulphate seemed to render the solution more palatable to the one sheep treated, and if the use of the mustard makes treatment easier, it may be recommended on this ground.

In default of scientific evidence we reserve judgment on the idea that mustard counteracts possible bad effects of copper sulphate on sheep.

ACKNOWLEDGMENT

Acknowledgment is due Dr. H. E. Moskey, of the Food, Drug and Insecticide Administration, who conducted the first of these tests.

REFERENCES

- ¹Branford, R.: Wireworm (*Haemonchus contortus*). Vet. Bul., Dept. Agr., Punjab, i (1920), 1.
²Hall, Maurice C., and Shillinger, Jacob E.: Miscellaneous tests of carbon tetrachlorid as an anthelmintic. Jour. Agr. Res., xxiii (1923), pp. 163-192.
³Kammlade, W. G., and McCulloch, E. C.: Skillful feeders can salvage "skip" lambs. 41st Ann. Rpt., Ill. Agr. Exp. Sta. (1927-1928), pp. 145-148.

As the Governor of North Carolina said to the Governor of South Carolina—Meet me in Detroit, in August.

LIGHTNING STROKE

By EARLE L. KITTRELL, Augusta, Ark.

Case 1: I was called to see a cow which the owner had just milked and fed in the barn. He went to the house and a few minutes later heard sounds of the cow struggling and kicking. He went back to the barn and found the cow down. He called me and, although I was only a few minutes in getting to the place, the cow was dead when I got there. I look around for evidence of poison and also for signs of lightning, as there had been a flash or two just recently. I thought I could smell hair burning and I made a diagnosis of death by lightning stroke. Autopsy indicated that I was right.

Case 2: I was called to a farm and found 20 hogs dead out of a herd of 45. The farmer who owned them said that he had fed them the night before and they were all right at that time. Upon inquiry I ascertained that the hogs had been under a tree in the center of the hog-lot. I decided that they must have been killed by lightning. Autopsy proved that I was correct in this case, too.

Case 3: Yesterday I was called to see a mare out in the center of a pasture. She was struggling and bleeding from a hole in the cranial cavity large enough for me to insert two fingers. Death was evidently caused by lightning. There have been quite a few other similar cases recently.

Detroit knows how to show you a good time.

AN ABNORMAL CASE OF PARTURIENT PARESIS

By C. H. NOFFSINGER, De Queen, Ark.

On May 23, 1929, I was called to see a Jersey cow, eight years old. The owner said that she had been ailing the day before. When I got there, at 9:00 a. m., I found the following: She was down and almost completely comatose. The head was stretched out, temperature 96° F., pulse almost imperceptible, dry hard feces in the rectum, pupils dilated, ears cold and very little milk in the udder.

Treatment: Inflation of the udder. I gave digitalin, $\frac{1}{4}$ grain; atropin sulphate, $\frac{1}{4}$ grain; and a rectal injection. I left two doses of a heart stimulant, one dose to be given each hour if she revived enough to take it. I returned at 2:30 and found her resting comfortably and chewing her cud. I removed the coverings from her and she immediately got to her feet and began eating. Then she walked over a quarter of a mile to the house and I then gave her a purge. She has been all right ever since.

Now, comes the abnormal part. This cow was fresh six months previously, to the day. Some practitioners claim that a cow will not have parturient paresis after four months, but I have had a number of cases, four to four and one-half months following calving. Some may say that I have been mistaken in my diagnosis, but, if it was such a mistake, why did I obtain the prompt recovery in this case? Results are what count and I got results and got them promptly. I would be pleased to hear from other veterinarians who have had similar cases.

BOVINE COCCIDIOSIS

By C. H. LARSON, Clarksville, Ark.

I was called to see a herd of 21 head (mixed) of dairy cattle. One cow about three years of age was dead. Another cow about the same age was sick and down. The temperature was subnormal and she was markedly emaciated. The membranes were distinctly pale and there was a history of bloody bowel discharges for about one week. Seven yearlings in the yard were sick and had temperatures ranging from 104° to 107° F. All were passing bloody feces and were off feed.

I immediately had the well animals moved to a hill pasture. The yard and so-called range is a veritable swamp. A postmortem examination of the dead animal revealed a hemorrhagic enteritis, with marked anemia, enlargement and cloudy swelling of the soft organs, and severe emaciation. This animal had been sick about ten days. I removed specimens of sections of the intestines and also took samples of the bloody bowel discharges from yearlings in the lot.

Microscopic examination of intestinal smears showed numerous coccidia and the lining epithelial cells revealed many of the parasites in various stages of development. Smears of the bloody feces showed myriads of coccidia. My diagnosis, of course, was coccidiosis. Treatment and control measures were instituted promptly.

MULTIPLE PREGNANCY IN A COW

By H. BUSMAN, Chicago, Ill.

Inspector-in-Charge, Bureau of Animal Industry.

As a matter of interest, I wish to report a case of multiple pregnancy in a cow slaughtered at establishment 521, the Illinois Packing Company, on June 11, 1929, there being six fetuses in the uterus.

The fetuses were normal in every respect, of uniform size and between the fifth and sixth months of development; two males and four females. They were in groups of three each, one group in each cornu. Each group was enclosed by a separate membrane.

While there are cases reported in veterinary literature of multiple pregnancy of six, or even more, fetuses found in the uterus, the case was of unusual interest to me, as I had not seen one with that many before, in all my experience on postmortem inspection.

ARMY VETERINARY SERVICE

CHANGES RELATIVE TO VETERINARY OFFICERS

Regular Army

The retirement, for age, of Colonel R. Vans Agnew, effective May 28, 1929, is announced.

Captain Frank C. Hershberger is relieved from further duty as transport veterinarian on the U. S. Army Transport "Meigs" and is assigned to duty at Fort Riley, Kansas, effective on or about August 28, 1929.

Captain Henry E. Hess has been relieved from duty at Madison Barracks, New York, and assigned to Fort Hamilton, New York, for duty.

Captain Everett C. Conant is relieved from duty at Fort Hamilton, New York, and assigned as transport veterinarian on the U. S. Army Transport "Meigs," effective on or about August 20, 1929.

Captain Frank H. Woodruff has been relieved from further duty at Fort Hoyle, Md., and will sail about August 20, 1929, for China.

Major Isaac O. Gladish is relieved from further duty at Fort Bliss, Texas, effective about August 10, 1929, and will sail on that date from San Francisco for the Panama Canal Department for duty.

Captain Samuel G. Kielsmeier is relieved from duty at Fort Bragg, N. C., and will sail from New York City on or about September 19, 1929, for duty in the Panama Canal Department.

Captain Herbert K. Moore will report for duty at Fort D. A. Russell, Wyoming, upon completion of his present tour of foreign service in China.

Captain Daniel H. Mallan has been granted leave of absence for one month and 19 days, effective on or about June 27, 1929.

Reserve Corps

Lt. Colonel Nelson Slater Mayo, Veterinary Reserve Corps, has been ordered to active duty, effective July 1, 1929, and will report to the Surgeon General, Washington, D. C., for training.

Promotions

Cook, Clarence John...to Captain.....1455 Lincoln St., Red Bluff, Calif.
Hicks, Jay Clyde.....to 1st Lt.....R-2, Tucson, Arizona.
Hughes, Arthur O.....to Major.....704 N. Penn Ave., Mangum, Okla.
Ryan, Charles L.....to 1st Lt.....7 Pleasant St., Dexter, Maine.

New acceptances

Beck, John D.....2nd Lt.....39th St. & Woodland Ave., Philadelphia, Pa.
Bendix, Wilmer L.....2nd Lt.....Danville, Va.
Bowman, Walter W...Capt.....Frederick, Okla.
Cross, Floyd.....Capt.....711 Matthews St., Ft. Collins, Colo.
Dowd, Eugene A.....Major.....339 Charles St., Boston, Mass.
Ellerman, Homer R...2nd Lt.....1817 Church St., Evanston, Ill.
Mosher, L. A.....Capt.....321 W. Spring St., Lima, Ohio.
Nimphius, H. F.....(N. G.) Capt...761 E. 163rd St., New York, N. Y.
Pease, Roy Herbert...2nd Lt.....Los Banos, Calif.

***If you remember the last meeting of the A. V. M. A. in
Detroit, you will not be found absent from the
Dynamic City, August 13-16.***

COMMENCEMENTS

ALABAMA POLYTECHNIC INSTITUTE

The commencement exercises of the Alabama Polytechnic Institute were held May 21, 1929. In the College of Veterinary Medicine the following graduates received the degree of Doctor of Veterinary Medicine:

C. R. Davis
J. B. Favara

J. G. Milligan
G. R. Kitchens

Let Detroit and Michigan hear you tell about your home town, August 13-16.

KANSAS STATE AGRICULTURAL COLLEGE

The sixty-sixth annual commencement exercises of the Kansas State Agricultural College were held at Manhattan, May 29, 1929. In the Division of Veterinary Medicine the degree of Doctor of Veterinary Medicine was conferred upon the following:

Carroll Ferdinand Alexander
Clair Lenna Butler
Frank Howard Callahan
Clifford Vernon Conger
Daniel DeCamp
Finis Ewing Henderson
Hugh Edward McClung

Ralph William Mohri
Needham Branch Moore, Jr.
Lawrence Orville Mott
Karl Willim Niemann
Charles Robert Omer
Harry Edward Schaulis
Francisco Rioja Taberner

Martin Van Der Maaten

The following graduates received commissions as second lieutenants in the Officers' Reserve Corps of the United States Army:

Daniel DeCamp
Hugh Edward McClure
Ralph William Mohri

Needham Branch Moore, Jr.
Lawrence Orville Mott
Charles Robert Omer

Karl Willim Niemann and Lawrence O. Mott were the honor students in the Division of Veterinary Medicine.

Dr. E. R. Frank (K. C. V. C. '24) received the degree of Master of Science.

The A. V. M. A. will take charge of the City of the Quick and the Dead, August 13-16.

STATE COLLEGE OF WASHINGTON

The commencement exercises of the State College of Washington were held June 3, 1929. Eighteen graduates from the College of Veterinary Medicine received the degrees of Bachelor of

Science in Veterinary Medicine and Doctor of Veterinary Medicine:

Edward Braun
Isidro Agustin
Roy Clarke
Jean Dirstine
Neil Halpin
Wilbur Kilpatrick
J. Karl Leaverton
Delphin Lumikaw
Robert McWherter

Allen Mills
Robert Morrison
Murrel Moys
Fred Richelieu
Charles Schroeder
Myron Thom
Ernest Willers
George Wright
Frank Whitcomb

Most of the 1929 class have accepted positions in the West or will go into practice in the Pacific Coast states.

Detroit is the home of the A. V. M. A., 716 Book Building.

COLORADO AGRICULTURAL COLLEGE

The commencement exercises of the Colorado Agricultural College were held in the college gymnasium, June 6, 1929. The commencement address was delivered by President Emeritus William O. Thompson, of Ohio State University.

In the Division of Veterinary Medicine the following graduates received degrees of Doctor of Veterinary Medicine:

William Harlan
Edward Howe

Jule Loftus
Charles Mueller

Lloyd Scrivner

With one exception, this is the smallest class in Veterinary Medicine to be graduated from the College.

The beautiful Book-Cadillac Hotel, Detroit, will be your home the week of August 13.

IOWA STATE COLLEGE

The commencement exercises at Iowa State College were unusual this year, on account of the celebration of the fiftieth anniversary of the inauguration of instruction in veterinary medicine at the institution. Alumni of the College began to gather on Friday evening, June 7, and a number of the classes held reunions. A considerable number had lunch together in the new Union Building at noon on Saturday.

At three o'clock, a program commemorating the founding of the Veterinary Division was started, with Dean Stange as master of ceremonies. He called upon President R. M. Hughes, who paid some very nice compliments to the Veterinary Division and

mentioned especially the high standing of the Division, on the Campus, and among the other divisions of the College.

Dr. J. H. McNeil, state veterinarian of New Jersey, who was professor and dean of the Veterinary Division at Ames from 1900 to 1908, was the next speaker. He emphasized the splendid type of young men who came to Iowa State College during his deanship. Dr. McNeil discussed the opportunities for young men in the veterinary field today and pointed out the developments that have taken place during the twenty-one years that he has been away from the institution.

Dr. David S. White, dean of the College of Veterinary Medicine, Ohio State University, was the next speaker and he presented a splendid paper, touching especially the educational developments and pointed out the needs for future developments that will be necessary in the field of veterinary education.

Among those who were present was Dr. D. C. Fairchild, who, together with Dr. Milliken Stalker, organized the Division of Veterinary Medicine. Dr. Fairchild was the first professor of pathology, histology and therapeutics and he spoke briefly of the early days of the College.

Dr. George C. Faville, of Hampton, Va., the first student to enroll in the Veterinary Division and the first graduate to receive the degree D. V. M. from Iowa State College, spoke in a reminiscent mood and his remarks were very interesting. Dr. Faville also spoke at the alumni banquet in the evening, which was attended by 600 friends and alumni of the College. After the banquet a smoker and get-together was held in the Union Building, at which a considerable number of veterinary alumni and their many friends were present.

On Sunday the baccalaureate sermon was delivered by Dr. E. F. Tittle, of Evanston, Ill. His theme was the influence of the machine upon the age in which we are living. It was very interesting.

In the afternoon, several hundred veterinary alumni and their friends visited the new veterinary research farm and from there they went to a reception at the home of President and Mrs. Hughes. The reception was held on the lawn and over 1000 people were present. The day was a delightful one and the alumni were in a very happy frame of mind. The reception was followed by a concert by the College Band.

On Monday the regular commencement exercises were held in the gymnasium, which was packed to capacity. Dr. C. P. Fitch,

of the University of Minnesota, gave the commencement address and also received the honorary degree Doctor of Science. President Hughes, in his remarks, referred a number of times to the Veterinary Division and its work.

The following received the degree of Doctor of Veterinary Medicine:

Adamson, Cecil T.	McCauley, Earl D.
Bailey, Westley W.	McCrillis, Harold L.
Bark, Hurdman W.	Mericle, Robert B.
Barry, James R.	Nilson, Walter L.
Beretta, Edward H.	Nordstrom, E. Harold
Black, James O.	Osborne, Harold
Bolin, Fonsoe M.	Smith, Edward D.
Carter, George S.	Stock, Leslie E.
Johnson, Percy C.	Tomshe, Emil J.
Lewis, Henry	Weber, Ralph E.

Walter L. Nilson was the honor man of the Veterinary Division and received the Judisch Scholarship Prize (\$25), providing membership in the A. V. M. A. and dues for three years. Mr. Nilson was also the highest honor *man* in the College. (A girl student in Home Economics had an average that was a fraction of one per cent higher than Mr. Nilson.)

You can get to the A. V. M. A. meeting in Detroit—you may not be able to go next year.

OHIO STATE UNIVERSITY

The fifty-second annual commencement exercises of the Ohio State University were held in the Coliseum at the State Fair Grounds, Tuesday, June 11, 1929. The commencement address was delivered by Charles F. Kettering, a graduate of the University, and now vice-president of the General Motors Corporation and president of the General Motors Research Corporation.

The College of Veterinary Medicine presented the following candidates for the degree of Doctor of Veterinary Medicine:

Frank Phifer Armfield	Clyde Lo Rayne Everson
Paul Bernard	Charles George Hall
Thomas Wilford Craver	William Arthur James
William Hezekiah Busic	Clifton Latshaw
Franklin Delaware Daughtrey	Emmet Karl LeDune
John Paul Delaplane	Henry Adrin Lidikay
	Cornelius Thibeault

The degrees were conferred by President George W. Rightmire of the University.

As the Governor of North Carolina said to the Governor of South Carolina—Meet me in Detroit, in August.

CORNELL UNIVERSITY

The annual commencement exercises at Cornell University were held June 17, 1929. The degree of Doctor of Veterinary Medicine was conferred upon the following:

Leon Abrevaya
Johanna Bertha Asmus
Donald Wyckoff Baker
Irwin George Bircher
Alton Parker Bouton
John Joseph Burgess
Guerino William Cangi
Ward Huntley Dwight
Ralph Theodore Ellison
James Lawrence Gibbons
Alexander Gow, Jr.
Francis Callis Greenman
Francesco Saverio Graziadei

Calvin Eugene Hall
*Charles Albert Ives
Samuel Augustus Johnson
Seth Darwin Johnson
George Edward McConnell
Peter Neil McCrank
Hugh Robertson Mouat
Jean Van Tassell Smith
Harry Clark Temple
Joseph Albert Thomas
Homer Gilbert Tully
Lemuel William Woodworth
William Wilson Wynn

The degree of Doctor of Veterinary Medicine was conferred upon Leo Anthony Fortune on February 6, 1929.

The following prizes were awarded for the academic year 1928-1929:

The Horace K. White Prizes

First Prize	Samuel Augustus Johnson
Second Prize	Lemuel William Woodworth

<i>The Hollingworth Honorarium</i>	Donald Wyckoff Baker
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The Jane Miller Prizes:

First Prize	Frank Bloom
Second Prize	Jesse Sampson

<i>The James Gordon Bennett Prize</i>	{ Calvin Eugene Hall
	{ Leon Abrevaya

<i>The Anne Besse Prize</i>	Irwin George Bircher
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*Died May 11, 1929.

Detroit knows how to show you a good time.

UNIVERSITY OF PENNSYLVANIA

At the commencement exercises of the University of Pennsylvania, held June 19, 1929, the degree of Doctor of Veterinary Medicine was conferred upon the following:

Richard Speier Bayard	Winslow Eaton Merrill
William Paul Doherty	James Albert Muffy
William MacDougall Lukens	Samuel Franklin Scheidy
James Vincent McCahon (cum laude)	Frank William Tarnow
Charles Edwin Watson	

The J. B. Lippincott Prize of \$100, for the highest general average for the entire four years of the course, the Jeannette Blair Prize of \$50, for the best work done in the Small Animal Clinic,

and the T. E. Munce Prize of \$25, for the highest general average in the courses in Animal Husbandry, were all awarded to James V. McCahon.

***Get right with the wife and children—take them to
Detroit, August 13-16***

MICHIGAN STATE COLLEGE

At the commencement exercises of the Michigan State College, held June 24, 1929, the following graduates in the Division of Veterinary Science received the degree of Doctor of Veterinary Medicine:

Eldon Clair Barclay	John Andrew Ellens
Harold John Buehler	Herbert C. Muth
Chester Frederick Clark	Walter W. Thompson
Stanley Clark Whitlock	

Since the 1928 commencement, the degree of Doctor of Veterinary Medicine was conferred upon Herman Dykema.

The senior prize offered by the Michigan State Veterinary Medical Association was awarded to John Andrew Ellens, who had the highest senior standing.

***The Detroit River in August is more beautiful than the
beautiful blue Danube.***

UNIVERSITY OF GEORGIA

The commencement exercises of the University of Georgia were held June 19, 1929. The Division of Veterinary Medicine conferred the degree of Doctor of Veterinary Medicine upon John Kolb Perry.

***The ideal convention city of the summer months—Detroit,
Michigan—welcomes the A. V. M. A., August 13-16.***

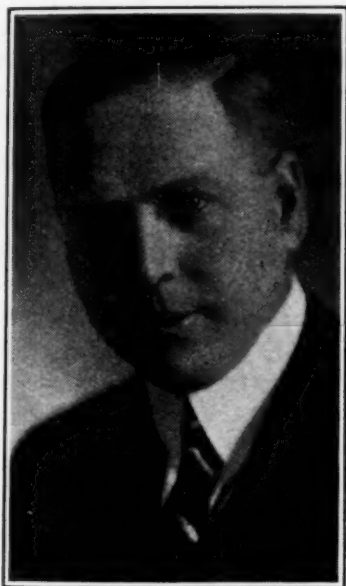
Manitoba Meeting

Dr. William Hilton, secretary-treasurer and registrar of the Veterinary Association of Manitoba, announces that Dr. W. F. Guard, of Amee, Iowa, will be the chief operator and demonstrator at the clinic to be held by the Association at Brandon, on July 5. A banquet will be held in the evening and the speakers will include Dr. Guard, Dr. J. B. Still, of Winnipeg, Dr. O. McGuirk, of Dauphin, and Dr. H. H. Ross, of Brandon.

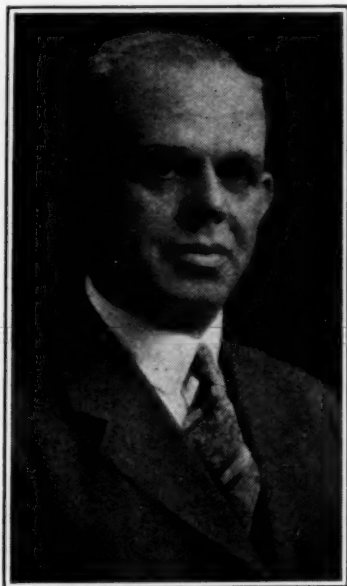
MISCELLANEOUS

Jensen-Salsbery Laboratories Reorganized

Announcement has been made of the re-organization of the Jensen-Salsbery Laboratories, Inc., following the withdrawal of Dr. H. Jensen from the firm, June 1. His interest has been acquired by Dr. C. E. Salsbery, Dr. G. G. Graham and Mr. A. K. Pearson. Dr. Salsbery and Dr. Graham have been actively identified with the organization ever since it was started, in 1915, and Mr. Pearson has been active in the business management of the concern for the past ten years. Dr. Graham is now Presi-



DR. G. G. GRAHAM



DR. C. E. SALSBERY

dent; Dr. Salsbery, Vice-President and Director of the Biological Laboratory; and Mr. Pearson, Secretary-Treasurer. Speaking for the new organization, President Graham states that the continuance of the management in the hands of the men who have been identified with the business since its inception assures rigid adherence to the merchandising policies that have been so largely responsible for the success of the company.

Flower Library Fund Increased

A gift of \$8,000, which will be added to the endowment of the Roswell P. Flower Library, has been presented to Cornell University, according to an announcement made recently by Dean Veranus A. Moore of the New York State Veterinary College. The Roswell P. Flower Library was established in 1897, for the use of the faculty and students of the New York State Veterinary College, by a gift of \$5,000 to Cornell University, from former governor Roswell P. Flower. The library fund was augmented in 1901 by a gift of \$10,000 from Mrs. Flower. Because of the acute situation resulting from the increased cost of publications, a faculty committee undertook to increase the endowment of the library fund. A committee consisting of Dr. Frank H. Miller, of New York, a trustee of the University, and two alumni, Dr. R. W. Gannett ('05), of Brooklyn, and Dr. Cassius Way ('05), of New York, collected the funds which have been turned over to the University through the Cornellian Council. It is the aim of the committee to raise \$2,000 additional, so that the library will have a permanent endowment of \$25,000. The income from the enlarged fund is to be used for the maintenance of the library as well as for the acquisition of additional volumes on veterinary and allied subjects.

The beautiful Book-Cadillac Hotel, Detroit, will be your home the week of August 13.

Tractor Sales

Wayne Dinsmore, secretary of the Horse Association of America, gives some interesting data in the production and sale of tractors in the United States for the past nine years.

<i>Year</i>	<i>Tractors Produced</i>	<i>Sold in United States</i>
1920	203,207	162,988
1921	73,198	
1922	99,692	101,192
1923	134,590	117,701
1924	120,643	99,011
1925	167,553	121,607
1926	181,995	130,753
1927	194,913	136,637
1928	171,137	99,491

Mr. Dinsmore points out that the number sold during 1928 was not enough to replace those worn out, even if the average life of a tractor is six years. Three years would be nearer to it.

International Veterinary Congress

The organization of the National Committee for the International Veterinary Congress has been completed. One of the functions of the Committee is to secure members to the International Congress from the United States.

The membership subscription is \$5.00. Members will receive all information regarding the Congress, as well as the complete proceedings, which will include not only all the papers and discussions presented but also the resolutions adopted in the different sections. The proceedings usually consist of three large volumes. It behooves every veterinarian to join the International Congress, whether he desires to attend or not, as the proceedings alone will more than repay him for the cost of membership.

Subscriptions should be sent to Dr. Adolph Eichhorn, Secretary of the National Committee, Pearl River, New York.

Visit one of the world's great automobile plants at Detroit.

Dr. Jensen in New Field

On another page in this issue of the JOURNAL, appears an announcement from Dr. H. Jensen, of Kansas City, directing attention to his retirement from the firm of Jensen-Salsbery, Inc., after thirty years spent in general practice, teaching and commercial work.

Dr. Jensen was graduated from the Chicago Veterinary College in 1900. For a number of years he was engaged in general practice at Weeping Water, Nebr. Later he joined the teaching staff of the Kansas City Veterinary College and held the chair of pharmacy, materia medica and therapeutics until about 1914. He was president of the Jensen-Salsbery Laboratories, Inc., from the time of its organization, in 1915, until his retirement from the firm, June 1.

Drive to Dynamic Detroit.

Complimenting Doctor Neuzil

The Blainstown (Iowa) Press, of May 17, 1929, carried an article under a three-column head, complimenting the Eastern Iowa Veterinary Association. A photograph of the officers of the organization featured the article. Dr. Paul V. Neuzil, who is secretary of the Eastern Iowa Veterinary Association, is one of Blainstown's distinguished citizens.

Idaho B. A. I. Men 100 Per Cent A. V. M. A.

Several months ago, a note appeared in the *JOURNAL* to the effect that the entire veterinary personnel of the Los Angeles County (Calif.) Live Stock Department was 100 per cent A. V. M. A. The number of veterinarians in the Department is eighteen and no one has accepted the challenge to show any other veterinary organization as large, with all members in the A. V. M. A.

This month we are pleased to direct attention to the fact that all veterinarians employed by the U. S. Bureau of Animal Industry and located within the state of Idaho are now members of the A. V. M. A. The staff consists of nine veterinarians as follows: Drs. W. A. Sullivan (McK. '07), A. J. Creely (San Fran. '15), A. S. Martin (K. C. V. C. '17), F. G. Miller (Iowa '08), E. G. Pigman (K. C. V. C. '13), J. E. Ellis (K. C. V. C. '03), F. H. Melvin (U. P. '20), W. E. Neary (Colo. '21), and W. C. Nye (Colo. '20).

Dr. Sullivan is Inspector-in-Charge of Tuberculosis Eradication and Hog Cholera Control in Idaho and he is also A. V. M. A. Resident Secretary for that state. It is a pleasure to publish this note directing attention to the fact that Dr. Sullivan's official family is 100 per cent A. V. M. A.

Cornell Students Drowned

A very sad accident occurred at Ithaca, N. Y., on May 11, when two veterinary students were drowned in Cayuga Lake. They were returning from a veterinary picnic which had been held down the lake. The boys apparently turned over in rough water and, although cries for help were heard by some of the cottagers along the lake, a boat could not be found in time to reach them. The two students drowned were Charles A. Ives, of Malverne, N. Y., a senior, and William W. Wagner, of Monroe, N. Y., a freshman. The bodies had not been recovered at the latest report. The water is very deep at the point where the drowning occurred and unless the water warms up considerably the recovery of the bodies is considered very doubtful.

Official Tour of Europe

In the August issue of the *JOURNAL* there will appear the itinerary of the tour being arranged in connection with the Tenth International Veterinary Congress, at London, Aug. 4-9, 1930.

ASSOCIATION MEETINGS

SOUTHEASTERN MICHIGAN VETERINARY MEDICAL ASSOCIATION

Although no reports of meetings of the Southeastern Michigan Veterinary Medical Association have appeared in the JOURNAL during the past few months, the organization has been quite active, particularly in view of the fact that the 1929 meeting of the American Veterinary Medical Association will be held in Detroit, in August.

The first fall meeting of the 1928-1929 season was held at the Hotel Statler, September 12, 1928. Drs. E. E. Patterson and A. S. Schlingman, of Detroit, rendered very interesting reports of the A. V. M. A. meeting held in Minneapolis during August. Dr. A. L. Tow, of Detroit, presented a very interesting paper, entitled, "Urinary Calculi in the Dog." Plans for the new building of the Michigan Humane Society were presented by the Secretary. This was followed by a very fine free discussion of the proper relations which should exist between local practicing veterinarians and humane societies.

OCTOBER MEETING

The regular monthly meeting was held at the Hotel Statler, October 10, 1928. The leading speaker of the evening was Mr. Frank G. Welbon, Jr., a prominent Detroit attorney, who spoke on the legal phases of veterinary practice. Mr. Welbon presented some very interesting information and his address was followed by a general discussion which was highly profitable to the veterinarians present. Dr. W. N. Armstrong, of Concord, Mich., president of the Michigan State Veterinary Medical Association, was the other speaker and he confined his remarks to the activities of the State Association. Attendance 27.

BANQUET

The November and December meetings were combined in the form of a banquet at the Hotel Statler, Wednesday evening, December 12, 1928. This was the first occasion in the history of the Association when ladies were invited to a formal banquet. There were forty-two veterinarians and wives present. The real purpose of the occasion was to start an organization to take care

of the many details in connection with entertaining the A. V. M. A. the following August. Dr. C. A. Taylor, of Detroit, president of the Association, introduced Dr. E. E. Patterson as chairman of the Entertainment Committee, who in turn introduced Dr. H. Preston Hoskins as toastmaster for the evening. The personnel of the Local Committee on Arrangements was announced and the chairmen of the various subcommittees were called upon to outline their plans as far as these had gone.

No meeting was held in January on account of a conflict in dates with the Veterinary Short Course at Michigan State College.

FEBRUARY MEETING

The meeting held February 13, 1929, at the Board of Health headquarters, was very unusual in that no formal program was arranged. Instead, a question-box was passed around and the members were invited to drop in questions. President Taylor called upon the members present to answer these questions as they were drawn out of the box and some of these proved to be so interesting that the meeting did not adjourn until about 11:30 p. m., the latest hour on record. Attendance 20.

MARCH MEETING

A regular monthly meeting was held at the Book-Cadillac Hotel, March 13, 1929. The program was opened by Dr. E. E. Patterson, who discussed plans for the Detroit meeting of the A. V. M. A., with particular reference to the clinic. Dr. J. R. McCarthy reported on the Work Horse Parade that had been held in Detroit recently. Dr. W. B. Whyte, of Highland Park, reported on the Detroit Dog Show. The program was concluded with a general discussion of the rabies situation in Detroit and vicinity. Plans for cooperating with the Detroit Board of Health were discussed. Drs. N. R. Boyes and O. C. Anderson were admitted to membership. Attendance 25.

APRIL MEETING

A regular monthly meeting was held at the Board of Health headquarters, April 10, 1929. Dr. J. R. McCarthy presented a very interesting case report, entitled, "Antepartum Paresis and Sequelae." Dr. Hugo Cornehl, Chief Veterinarian, Detroit Department of Health, presented a very interesting paper, entitled, "Municipal Meat Inspection in the City of Detroit." He was followed by Dr. R. F. Vermilya, B. A. I. inspector-in-

charge at Detroit, who presented a very interesting paper describing the various activities of the U. S. Bureau of Animal Industry. This was followed by an exhibit of pathological specimens from food-producing animals. This exhibit was prepared and shown by Dr. E. F. Jameson, of the local B. A. I. staff.

The annual election of officers resulted as follows: President, Dr. C. W. Eddy, Detroit; vice-president for Wayne County, Dr. L. H. LaFond, Detroit; vice-president for Macomb County, Dr. L. H. Smith, Mt. Clemens; vice-president for Oakland County, Dr. R. H. Wilson, Rochester; secretary-treasurer, Dr. H. Preston Hoskins, Detroit. Attendance 28.

MAY MEETING

The regular monthly meeting was held at the Book-Cadillac Hotel, May 8, 1929. This meeting was a rather unusual one in that it was attended by all the members of the A. V. M. A. Committee on Program, who had been in session during the day. Members of the Committee were called upon for short talks.

Dr. F. H. Brown, state veterinarian of Indiana, spoke very feelingly of the obligations which veterinarians owe to their profession and particularly to the older members of the profession who laid such a firm foundation for veterinary medicine. Dr. Brown said that it would be a very fine thing if the veterinarians of today would show the same willingness to make personal sacrifices in behalf of the profession that were characteristic of veterinarians who were the leaders in the profession twenty or thirty years ago.

Dr. W. A. Hagan, of the New York State Veterinary College at Cornell University, confined his remarks to tuberculin and the tuberculin tests. He spoke about other acid-fast infections, the skin lesions encountered in cattle and non-specific reactions obtained with tuberculin.

Dr. H. J. Milks, New York State Veterinary College at Cornell University, spoke on canine distemper and briefly reviewed the results of the investigations conducted during recent years at Mill Hill near London, England. He explained the Dunkin-Laidlaw method of immunizing against canine distemper.

Dr. B. Scott Fritz, Pennsylvania Bureau of Animal Industry, Harrisburg, Pa., spoke on the Pennsylvania plan of handling infectious abortion. He explained that there are now 33,000 animals in 2300 herds in Pennsylvania operating under the plan.

Dr. R. O. Biltz, Pennsylvania Bureau of Animal Industry, Harrisburg, Pa., spoke on poultry disease control in Pennsylvania. He explained the work being done in the Keystone State in an effort to control poultry diseases and emphasized the importance of the practicing veterinarian in this connection.

A rising vote of thanks was extended the visiting veterinarians for supplying the program for the evening. Attendance 34.

JUNE MEETING

The regular monthly meeting was held at the Board of Health headquarters, June 12, 1929. Dr. L. H. LaFond gave some interesting case reports and started a very interesting discussion on the proper diet for dogs under different conditions. One very important point that was stressed by Dr. LaFond was the desirability of having more agreement among veterinarians as to the proper diet for dogs in order that there may in turn be less confusion in the minds of our clients.

Dr. F. D. Egan spoke at considerable length concerning legislation against the ear-cropping of dogs. Dr. Reuben Hilty, of Toledo, Ohio, former president of the A. V. M. A., made a very strong plea for better cooperation between veterinarians and humane societies. He briefly reviewed what the A. V. M. A. had accomplished along this line during the past few years. Attendance 27.

The Association decided to recess during July and August and resume meetings in September.

H. PRESTON HOSKINS, *Secretary*

Let Detroit and Michigan hear you tell about your hometown, August 13-16.

SAGINAW VALLEY VETERINARY MEDICAL ASSOCIATION

The spring meeting of the Saginaw Valley Veterinary Medical Association was held in the auditorium of the County Agriculturist's office, Saginaw, Mich., April 11, 1929. There was a record attendance at the meeting, about forty veterinarians being present. This is quite remarkable in view of the almost impassable condition of the roads at the time. Some of those present traveled as far as 100 miles to attend.

Dr. F. M. Lambie, of Midland, discussed "Traumatic Pericarditis in Cattle." He stressed the economic importance of an

early diagnosis for salvage purposes. He also dwelt on the ever-increasing number of cases of this condition.

Dr. E. T. Hallman, Michigan State College, discussed the control of abortion disease in a very thorough manner. He emphasized the importance of the serological tests for locating infected animals so that these might be isolated. He advocated the frequent testing of infected herds and also pointed out the great financial loss sustained by cattle-owners throughout the United States from this disease. Dr. Hallman said that it was greater than the loss caused by bovine tuberculosis.

Dr. F. A. Burlingame, of Chesaning, discussed "Municipal Meat Inspection from the Standpoint of Public Health." He mentioned the increasing number of calls he has been receiving from farmers in his territory asking for advice on animals slaughtered for home consumption.

Dr. H. L. Cole, of Saginaw, discussed "Internal and External Parasites and Their Treatment." He described the technic for microscopic diagnosis of various parasitic diseases. He had several specimens prepared on slides and these were placed under the microscope for the convenience of those present.

Dr. B. J. Killham, State Veterinarian, discussed the rabies situation in different parts of the State and made suggestions for the better control of the disease.

The next meeting of the Association will be held in the fall at Chesaning and the program committee is planning a clinic in conjunction with the meeting.

MICHAEL D. DUCEY, *Secretary*

Michigan is the summer playground of America—Detroit is the gateway.

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The regular monthly meeting of the Veterinary Medical Association of New York City was held at the Academy of Medicine Bldg., 103rd St. and 5th Ave., New York City, Wednesday evening, May 1, 1929.

The speakers of the evening were Dr. Emmett W. Price, Bureau of Animal Industry, Washington, D. C., and Dr. Merriam Rodgers, Brooklyn, N. Y.

Dr. Price spoke on parasites. He stated that it has been estimated that in some veterinarians' practices the cases of parasitism

in dogs and cats averages about 50 per cent. Dr. Price stated that of the external parasites *Demodex folliculorum* is the most difficult one to combat. He recommended the following prescription:

Kerosene.....	8 ozs.
Linseed oil.....	8 ozs.
Phenol.....	1 oz.
Oil of tar.....	1 oz.
Sulphur.....	4 ozs.

This preparation should be applied to one-fourth of the body at a time. Extreme care should be taken when using it on small delicate dogs. Another preparation for small dogs is as follows:

Balsam of Peru
Alcohol
Soft soap, equal parts, 4 ozs.

For cats, sulphur ointment is best; eight parts of lard to one of sulphur. For ear mites, use chloroform, one part, and castor oil, nine parts. For fleas in cats, dusting powders should be used in preference to the coal-tar products.

Dr. Price stated that the reason why puppies have worms at such an early age is because the larvae filter through the placental membranes. For fecal examinations the salt flotation method is recommended. Pour hot water over a quantity of salt and make a saturated solution. Place a small amount of the feces in a wide-mouth bottle and half fill with salt solution. Stir well. Then fill to the top with the salt solution. Place a slide over same and remove in about fifteen minutes. Worm eggs will be found on the slide. This method is not so good for tapeworms and flukes but is excellent for roundworms, hookworms or whipworms.

Oil of chenopodium is best for roundworms, according to Dr. Price, but care should be used in administering the drug to devitalized dogs and it should always be followed with castor oil. Tetrachlorethylene also is excellent. Santonin is very useful for dogs in a weakened condition, as it is not a gastro-intestinal irritant in normal dosage. In all cases of dogs that have been in the South, it is well to make an examination of blood smears for evidence of filaria.

Dr. Merriam Rodgers was the next speaker. Dr. Rodgers has specialized in ophthalmology for many years and he gave a very interesting and instructive talk on the eye and its appendages. For local anesthesia of the eye, he recommended holocain 1 per cent or butyn. For pus in the eye, examine the pus micro-

scopically, staining with methylene blue. For pneumococcus infection of the eye, optochin, 1 per cent, t. i. d., is indicated and gives excellent results. It is necessary to stimulate the patient occasionally by using injections of boiled milk which increase the number of white blood cells.

Antiseptics used in eye conditions should be applied in the following strengths: Argyrol, 30 per cent; mercurochrome, 2 per cent; neosilvol, 10 per cent.

For ectropion, cauterize the conjunctiva in eight or nine places. In cases of pterygium, it is best to dissect and turn back. Excluding injuries, most eye lesions are due to systemic disturbances.

In lime burns of the eye, use no water. Ammonium chlorid, 1 per cent, acts as a chemical antidote. Glaucoma is usually due to systemic infection. In treating incipient cataracts, a small quantity of cyanid of mercury, 1-6000 solution, should be injected in the conjunctiva.

A general discussion followed Dr. Rodgers' talk and a rising vote of thanks was extended to him and Dr. Price for their contributions to the program.

R. J. GARBUTT, *Secretary.*

Visit one of the world's great automobile plants at Detroit.

HUDSON VALLEY VETERINARY MEDICAL SOCIETY

The regular quarterly meeting of the Hudson Valley Veterinary Medical Society was held at Catskill, N. Y., May 8, 1929. The members and guests had luncheon at the Hotel Saulpaugh and then repaired to the well-equipped hospital of Dr. L. L. Parker, where the afternoon program took place with Dr. Wm. Henry Kelly, of Albany, in the chair.

Dr. Parker recently installed a very complete x-ray machine with fluoroscope attachment and the advantages of this instrument were explained and demonstrated by an expert present.

Dr. W. W. Williams, of Springfield, Mass., then addressed the gathering and gave some very interesting observations and experiences with respect to sterility and allied troubles. This was followed by an extensive question-box in which nearly all members present took part.

Questions on abortion were discussed and references made to the use of various feeding rations as factors in controlling breeding difficulties. The use of minerals to counteract other deficiencies

was considered and the speaker pointed out that in some instances the animal system was not capable of making use of these ingredients even though contained in the feed and, when such conditions existed, attention should first be given to remedying the defect in the body mechanism.

The attendance was 37.

J. G. WILLS, *Secretary.*

The Southeastern Michigan Veterinary Medical Association, with headquarters at Detroit, claims to be the liveliest local veterinary association on the map.

CONESTOGA VETERINARY CLUB

The regular monthly meeting and the sixteenth annual Shad Supper of the Conestoga Veterinary Club was held at the Stock Yards Inn, Lancaster, Pa., May 9, 1929.

The meeting was called to order at 5:00 p. m. and started with a splendidly illustrated lecture by Dr. H. E. Bemis of the University of Pennsylvania School of Veterinary Medicine, entitled, "Personalities of Some Distinguished European Veterinarians." Pictures of the buildings, their equipment and the personnel of the institutions visited by Dr. Bemis on his European tour last year were shown. This lecture was under the auspices of the University of Pennsylvania Veterinary Extension, directed by Dr. G. A. Dick, and it was particularly gratifying to those present. It made us feel personally acquainted with the kind, friendly and distinguished veterinarians of Continental Europe.

Dr. R. C. Gross, of Elizabethtown, president of the Club, acted as toastmaster. The first speaker was Hon. F. C. Musser, mayor of Lancaster and president of the Lancaster Live Stock Exchange. He spoke briefly concerning a proposed ordinance for local meat inspection and pointed out the fine work that veterinarians do in the community. Hon. A. B. Hess, speaker of the Pennsylvania House of Representatives, was the next speaker. He paid the veterinary profession a number of very nice compliments.

The nine veterinarians constituting the staff of the Pennsylvania B. A. I. Laboratory were in attendance and Dr. M. F. Barnes, Director, described an epizootic affecting deer in Clearfield County and a quarantine which has been placed on a large importation of Hungarian partridges. Among the other speakers were Dr. J. B. Reidy, Harrisburg; Dr. H. B. Mitchell, Lancaster;

Dr. J. D. Jones, Harrisburg; Dr. J. B. Rynkiewitz, New Bloomfield; Dr. E. L. Cornman, Marietta; Dr. R. O. Whipple, Lemoyne; and County Agent F. S. Bucher.

We missed Dr. T. E. Munce, president of the A. V. M. A., who found it impossible to be present at the last moment. The supper was a splendid one and was heartily enjoyed by the 59 members and their friends who were present.

H. S. WEBER, *Secretary*

Detroit is the home of the A. V. M. A., 716 Book Building.

NORTH CENTRAL IOWA VETERINARY MEDICAL ASSOCIATION

The annual meeting of the North Central Iowa Veterinary Medical Association was held at the Wahkonsa Hotel, Fort Dodge, May 27, 1929. The attendance was larger than has been the case in the past two years. Much interest was shown in the papers presented. Dr. T. M. Rossing, of Bode, presided.

At the business session the following officers were elected for the ensuing year: President, Dr. B. E. McCulloch, Eagle Grove; vice-president, Dr. J. J. Williams, Fort Dodge; secretary and treasurer, Dr. H. J. Shore, Fort Dodge.

The literary program was as follows:

"Tuberculosis Eradication in Iowa," by Dr. J. A. Barger, Des Moines.

"A Message to Practicing Veterinarians," by Mr. Geo. Judisch, Ames.

"Diseases of Swine," by Dr. J. O. F. Price, Algona.

"Some Observations on Canine Practice," by Dr. A. Kaderabek, Fort Dodge.

The paper presented by Dr. Price was very freely discussed and among those who participated in the discussion were Dr. C. N. McBryde, of the Bureau of Animal Industry, Ames, Iowa, and Dr. C. G. Cole, assistant to Dr. McBryde.

At 6:30 p. m., the Fort Dodge Serum Company tendered a dinner dance for the members of the Association, their families and friends.

H. J. SHORE, *Secretary*

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The line forms at the right, in Detroit,
August 13-16, led by the A. V. M. A.***

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At the business session the following officers were elected for the ensuing year: President, Dr. B. E. McCulloch, Eagle Grove; vice-president, Dr. J. J. Williams, Fort Dodge; secretary and treasurer, Dr. H. J. Shore, Fort Dodge.

The literary program was as follows:

"Tuberculosis Eradication in Iowa," by Dr. J. A. Barger, Des Moines.

"A Message to Practicing Veterinarians," by Mr. Geo. Judisch, Ames.

"Diseases of Swine," by Dr. J. O. F. Price, Algona.

"Some Observations on Canine Practice," by Dr. A. Kaderabek, Fort Dodge.

The paper presented by Dr. Price was very freely discussed and among those who participated in the discussion were Dr. C. N. McBryde, of the Bureau of Animal Industry, Ames, Iowa, and Dr. C. G. Cole, assistant to Dr. McBryde.

At 6:30 p. m., the Fort Dodge Serum Company tendered a dinner dance for the members of the Association, their families and friends.

H. J. SHORE, *Secretary*

***Michigan has the greatest coast-line of any state.
The line forms at the right, in Detroit,
August 13-16, led by the A. V. M. A.***

NECROLOGY

S. H. GILLILAND

Dr. Samuel Howard Gilliland, of Marietta, Pa., died in St. Joseph's Hospital, Lancaster, Pa., May 27, 1929, after an extended illness.

Born at Oak Hill, Centre County, Pa., June 10, 1877, Dr. Gilliland was educated in the local public schools, Bellefonte Academy, Millersville State Normal School and the University of Pennsylvania. From the latter institution he received the degree of Doctor of Veterinary Medicine in 1901 and the degree of Doctor of Medicine in 1904. While pursuing his studies at the University of Pennsylvania, he specialized in bacteriology and worked in the laboratory of the Pennsylvania Live Stock Sanitary Board from 1898 to 1905. In 1904 he was made director of the laboratory, succeeding Dr. M. P. Ravenel, who severed his connections with the Pennsylvania State Live Stock Sanitary Board at that time to become associated with the Henry Phipps Institute for the Study and Prevention of Tuberculosis, located in Philadelphia.

Dr. Gilliland was instructor in veterinary bacteriology and laboratory diagnosis at the University of Pennsylvania in 1901 and 1902. In conjunction with the late Dr. Leonard Pearson and Dr. M. P. Ravenel, he conducted and published the results of a large number of experiments upon the production of immunity against tuberculosis in animals. On account of ill health, he severed his relations with the Pennsylvania State Live Stock Sanitary Board in 1906 and accepted the presidency and office of Director of Laboratories of the firm of H. M. Alexander & Company, producers of biological products, at Marietta, Pa.

Following the death of Dr. Leonard Pearson, in 1909, Dr. Gilliland was appointed by Governor Stuart as State Veterinarian of Pennsylvania and Secretary of the State Live Stock Sanitary Board, which positions he held until 1911, when he was obliged to decline reappointment on account of poor health.

In 1912 and 1913, Dr. Gilliland was Director of the Laboratories of the Pennsylvania State Department of Health, under the supervision of the late Samuel C. Dixon, Commissioner of Health of Pennsylvania. Dr. Gilliland retained the office of president of H. M. Alexander & Company until 1916, when the

company was merged with the Gilliland Laboratories, Inc., of which organization he was president until 1929. During the past two years, although he still remained as president, his health was such that he was unable to conduct the affairs of the company actively.

Dr. Gilliland joined the A. V. M. A. in 1903. He was a fellow of the American Public Health Association, a member of the American Medical Association, Philadelphia Pathological Society, Philadelphia Medical Society, University Club of Philadelphia, the Medical Club of Philadelphia, Medical Club of Lancaster, University of Pennsylvania General Alumni Society, Hamilton Club of Lancaster, National Tuberculosis Association, Pennsylvania State Veterinary Medical Association, United States Live Stock Sanitary Association and the Conestoga Veterinary Club.

E. L. C.

W. H. COREY

The Maine Veterinary Medical Association adopted the following resolution on the death of Dr. W. H. Corey, of Newport, Maine, at the regular quarterly meeting held October 10, 1928:

WHEREAS, The Supreme Architect of the universe has seen fit to remove from our midst Dr. W. H. Corey, an active and highly esteemed member of this Association, therefore be it

Resolved, That we are profoundly impressed with a deep sense of bereavement at the loss of our friend and associate, and hereby tender to the members of his stricken family our heartfelt sympathy in this great affliction, and these resolutions be spread upon the records and a copy sent to his family.

Dr. Corey was born at Deansboro, N. Y., October 25, 1861, and was a graduate of the Ontario Veterinary College, class of 1898. He joined the A. V. M. A. in 1921.

WILLIS G. BENNER

Dr. Willis Grant Benner, of the U. S. Bureau of Animal Industry, who resided in Washington, D. C., died March 20, 1929, following an illness of about six weeks. Dr. Benner, who was 61 years of age, was born in South Whitehall, Pa. He was graduated from the Ontario Veterinary College in 1890 and was engaged in general practice at Doylestown, Pa., until the latter part of 1902. when he entered the Bureau service. For the past 19 years, Dr. Benner had been identified with tuberculosis eradication work. His assignments included the District of Columbia

and vicinity, New England and California. Since September, 1925, he had been working on tuberculosis eradication in Maryland.

Dr. Benner was one of the pioneers in tuberculosis eradication and was always considered a very efficient, capable and energetic worker. He is survived by his widow, two daughters and four sons.

A. E. W.

ROY C. FROST

Dr. Roy C. Frost, formerly of Washington, D. C., died in the sanitarium at Blue Ridge Summit, Md., May 19, 1929. He was a native of Maryland, born May 26, 1886. He was a graduate of the United States College of Veterinary Surgeons, class of 1914. For a time following his graduation he was in the employ of the National Vaccine Company, of Washington, D. C., and later with the District Health Department. In 1924, he entered the employ of the U. S. Bureau of Animal Industry and was assigned to the Pittsburgh, Pa., station on meat inspection. More recently he had been stationed at Baltimore, Md.

Dr. Frost joined the A. V. M. A. in 1915. He served in the U. S. Navy for four years. He is survived by his widow, one daughter and two sisters. Interment was at Arlington Cemetery.

H. R. ESTES

Dr. H. R. Estes, of Brandon, Manitoba, died at the General Hospital, Brandon, May 25, 1929, after a lingering illness.

A native of New York State, Dr. Estes received his early education at local schools and then decided to enter the Ontario Veterinary College. He was graduated in 1912 and entered the service of the Health of Animals Branch, Dominion Department of Agriculture, and for a time was stationed at Winnipeg and later at Brandon. He remained at the latter point for eight years in charge of tuberculosis eradication work in that district under the Municipal Tuberculosis Order.

Dr. Estes was a member of the Veterinary Association of Manitoba, the Professional Institute of the Civil Service of Canada, the Amalgamated Civil Servants and the First Presbyterian Church. He is survived by his widow and one daughter. Interment was at Owen Sound, Ont.

W. H.

CHARLES W. SIMPSON

Dr. Charles W. Simpson, of Williamston, Mich., died January 29, 1929. He was a graduate of the Grand Rapids Veterinary College, class of 1904, and practiced at Webberville, Mich., for a number of years before locating in Williamston. He was a member of the Michigan State Veterinary Medical Association.

DOLPH S. DE WOLF

Dr. D. S. DeWolf, of Hart, Michigan, died June 1, 1929, of heart disease.

Born in Grand Rapids, Mich., Dr. De Wolf located in Hart following his graduation from the Ontario Veterinary College in 1900. He was in active practice up until about five years ago when he became associated with the Kunkel Manufacturing Co. He was a very capable veterinarian and was highly esteemed by his colleagues. He is survived by his widow, two sons, three brothers and one sister.

He was a member of the Michigan State Veterinary Medical Association.

ENOS S. MOYER

Dr. Enos S. Moyer, of Perkasio, Pa., died May 30, 1929, at the age of 58. He was a graduate of the New York College of Veterinary Surgeons, class of 1892, and had practiced in Bucks County, Pennsylvania, and vicinity ever since his graduation. He was a member of the Pennsylvania State Veterinary Medical Association.

AMOS WINEGAR

Dr. Amos Winegar, of Howell, Michigan, died June 15, 1929, following injuries received when his automobile was struck by another car at one of the main crossings in Howell. He was 78 years of age. He was a registered non-graduate and practiced in Howell for 50 years.

GEORGE W. COPPES

Dr. George W. Coppes, of Ligonier, Indiana, died June 9, 1929, following a long illness, at the age of 73. He was a graduate of the Ontario Veterinary College, class of 1879, and practiced in Ligonier and vicinity for 50 years.

Our sympathy goes out to Dr. Clarke Hedley, of Springfield, Mass., in the death of his daughter, Mrs. Mozelia Hedley Russ, of Harrisburg, Pa., of embolism following an operation; to Dr. Bernard P. Wende, of Buffalo, N. Y., in the death of his wife, after a protracted illness; to Dr. and Mrs. N. S. Mayo, of North Chicago, Ill., in the death of their daughter, Dorothy, at St. Lukes Hospital, Chicago, May 30, 1929; and to Dr. B. M. Underhill, of Media, Pa., in the death of his mother, Mrs. Sarah Pearson Underhill, at Media, recently, at the age of 100.

PERSONALS

MARRIAGES

Dr. Alfred Savage (Corn. '14) to Miss Mary Norquay, both of Winnipeg, May 21, 1929, at St. Andrews, Man.

Dr. L. E. Case (Corn. '08) to Miss Lois Condon Stegman, both of Honolulu, Hawaii, June 4, 1929, at Honolulu.

BIRTHS

To Dr. and Mrs. C. A. Henley, of New Berlin, Ill., a son, Robert Wayne, May 26, 1929.

To Dr. and Mrs. R. S. Betts, of Lost Nation, Iowa, a son, Kenneth George.

To Dr. and Mrs. E. C. Deubler, of Philadelphia, Pa., a daughter, June 12, 1929.

PERSONALS

Dr. W. H. Chivers (Iowa '28) has located at Manson, Iowa.

Dr. H. L. Messmore (Chi. '14) has located at Oneida, Ill., for general practice.

Dr. E. J. Jelden (K. S. A. C. '22) has located for general practice at Columbus, Nebraska.

Dr. L. N. Brown (Ont. '29) has located for practice at Auburn, Me. Address: 441 Turner St.

Dr. R. T. Gregory (McK. '18) has changed locations from Newport News, Va., to Fredericksburg, Va.

Dr. H. E. Burdick (Ont. '26) has opened a dog, cat and bird hospital at 12234 Linwood Ave., Detroit, Mich.

Dr. T. J. Eagle (K. C. V. C. '03) sends us a change of address from Maryville, Mo., to Savannah, Mo., Route No. 3.

Dr. G. S. Harshfield (O. S. U. '26) reports a change of address from Canton, Ohio, to Warren, Ohio, R. F. D. No. 5.

Dr. C. A. Sayre (O. S. U. '14) has changed locations from Wauseon, Ohio, to London, Ohio. Address: 93 Elm St.

Dr. F. A. Laird (Chi. '02) has tendered his resignation as Chief Veterinarian, Illinois State Department of Agriculture.

Dr. D. W. Gates (U. P. '25) has removed from Bellefonte, Pa., to Bridgeport, Pa. Address: 508 Eastburn Terrace.

Dr. T. S. Leith (Iowa '14) reports a change of address from DeFuniak Springs, Fla., to Oskaloosa, Iowa, Route 1.

Dr. George M. Carson (McK. '12) has removed from Thompson, Iowa, to Albert Lea, Minn. Address: 705 Minnesota St.

Dr. Lyle H. Briggs (Mich. '26) resigned from the U. S. B. A. I. service June 1. He was stationed in Chicago on meat inspection.

Dr. J. B. Way (K. C. V. C. '14), of Louisville, Ky., will move into his new home on July 8. His new address is 304 Pleasant View.

Dr. J. A. Ford (Ont. '10), formerly of Los Angeles, Calif., is now conducting the practice of the late Dr. W. L. Greening, of Long Beach.

Dr. D. K. Collins (O. S. U. '26) is at present employed by Dr. L. I. Beller, (St. Jos. '18), at 10316 Long Beach Bldg., Huntington Park, Calif.

Dr. H. J. Satorius (Chi. '11), who has been in general practice at Middletown, Ill., for about eighteen years, has removed to Mason City, Ill.

Dr. L. A. Wileden (Mich. '13), of Mason, Mich., is laid up with a broken leg, just below the knee. He was castrating a colt when the accident occurred.

Dr. Maurice C. Hall (Geo. Wash. '16) has been seriously ill at the Walter Reed Hospital, Washington, D. C. The latest report is that he is convalescing.

Dr. S. R. Johnson (K. S. A. C. '20), formerly of Lansing, Mich., has removed to Kansas City, Mo., where he is associated with Ashe Lockhart, Inc.

Dr. C. L. Woolard (Chi. '17) has been reappointed Montgomery County (Ill.) Veterinarian at a salary of \$3600 and an allowance of \$400 per year for expenses.

Dr. Earl M. Keef (San Fran. '13) has resigned his position with the State Department of Agriculture and has accepted a position with the Fuller Rancho, Corona, Calif.

Dr. John R. Martell (Ont. '28) is now associated with Dr. G. F. Ewalt (Ont. '14), at the Grand River Veterinary Hospital, 10117 Grand River Ave., Detroit, Mich.

Dr. T. H. Ruth (Chi. '06), of De Smet, S. Dak., has been appointed State Veterinarian, succeeding Dr. M. W. Ray (K. C. V. C. '11). Dr. Ruth has moved to Pierre.

Dr. D. E. Westmoreland (Ird. '04), state veterinarian of Kentucky, was injured in an automobile accident on June 5, on the Louisville Highway about twelve miles west of Frankfort.

Dr. F. E. Henderson (K. S. A. C. '29) has accepted an appointment as Live Stock Inspector with the Nevada State Live Stock Sanitary Board. He will have his headquarters at Elko, Nevada.

Dr. Karl W. Niemann (K. S. A. C. '29) has accepted a position in the Department of Veterinary Science, University of Nevada, at Reno. He will be engaged in research work with animal diseases.

Dr. Benj. McInnes (R. C. V. S. '74) and his son, Dr. B. Kater McInnes (U. P. '11) served in the capacity of official veterinarians to the fourth annual horse show held in Charleston, S. C., April 5 and 6.

Dr. I. D. Wilson (Iowa '14) has been granted a sabbatical leave of absence for one year by the Virginia Polytechnic Institute and he expects to spend the year studying parasitology at Iowa State College.

Dr. M. E. Howell (St. Jos. '16), formerly of Sioux Falls, S. Dak., and associated with the Allied Laboratories, Inc., has been transferred to the Kansas City territory. Dr. Howell gives his new address as 808 Lane St., Topeka, Kans.

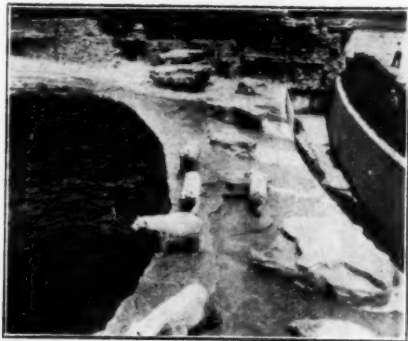
Dr. C. E. Turnbull (Gr. Rap. '10), who has been in the employ of the U. S. Bureau of Animal Industry for a number of years, recently resigned and has gone into general practice in Detroit, Mich., in partnership with Dr. A. L. Tow (Ind. '18).

Dr. George R. White (Columbia '97), of Nashville, Tenn., has been appointed a member of the State Board of Veterinary Medical Examiners, succeeding Dr. Wm. M. Bell (K. C. V. C. '92), of Nashville, whose term recently expired.

Dr. J. A. Allen (Ont. '16), Winnipeg, Man., specialist in fur-bearing animals, and Director of Health of the All Star Fox Ranch, Winnipeg, left recently to visit the All Star Fox Ranch at Pine Knot, Calif. He expected to be away from Winnipeg for several weeks.

Dr. F. W. Lupfer (Chi. '07), of Galva, Ill., was a hospital patient recently as the result of getting a hold of the wrong bottle when he went to his medicine chest to get a dose of cough medicine. Instead he took a bottle containing acid and badly burned his throat as a result.

Dr. Bernard Johnson (O. S. U. '11), of Spokane, Wash., started for St. Joe, Idaho, in an aeroplane, on May 16, to search for a Spokane citizen reported missing near St. Joe. Dr. Johnson was accompanied by a second plane, and the two craft carried a pair of trained German police dogs owned by Dr. Johnson, to be used in the search.



Scene at Detroit Zoo